NOTES
ON
PATHOLOGY

GENERAL PATHOLOGY.

J. Ryland Whitaker.

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BY

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TO

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OF THE ROYAL COLLEGE OF SURGEONS, EDINBURGH.

This Little Work is Dedicated

IN TOKEN OF LONG AND FIRM FRIENDSHIP, AND WITH

MUCH RESPECT AND ESTEEM.

PREFACE.

THIS little book pretends to no more than its title indicates—viz., a series of NOTES ON PATHOLOGY. It is not intended as a Text-book of the subject, but merely as a guide to the Student in attending Lectures or in reading any of the standard works on Pathology, such as Woodhead, Zeigler, Coats, etc. The theories and ideas advanced will in many cases be found to reflect the teachings of the Edinburgh School on the point at issue, for it was under the able guidance of its teachers that the author gathered the first rudiments of the Science.

THE AUTHOR.

EDINBURGH, April 1890.

CONTENTS.

Section II.

INFLAMMATION AND ITS RESULTS.

Section III.

ALTERED CONDITIONS OF NUTRITION.

RETROGRESSIVE CHANGES.

PROGRESSIVE CHANGES.

Section IV.

PARASITES AND PARASITIC DISEASES.

INTRODUCTION.

I. **Definition.**—Pathology has been defined as "*the Science of Disease*"—*i.e.*, that Science which treats of the causes, the development, and the results of disease. To understand this definition we require to have a clear idea of the meaning of the term—disease. Now, *Disease* has been defined as "a complex of some deleterious agency acting upon the body, and of the phenomena—actual and potential—due to the operation of that agency" (Bristowe). The meaning of this definition is that disease is a deterioration in, or a deviation from, what, by long experience, we have come to regard as *Health*—that is, it is a deterioration in the performance of some or all of the functions of the body, as the result of the action of agents which are injurious either in their nature or in their effects.

Health is subject to certain chemical, physical, and biological laws, and so is disease; and hence, Pathology may be regarded as abnormal Physiology; and like that Science is based upon the cellular theory, which teaches that all organs and organisms consist of cells and cell aggregates, or of modifications thereof.

At present, Pathology is a purely experimental science, for we find certain signs in the affected part before death, and certain more or less marked changes after death; and it is the province of Pathology to investigate the connection between these two sets of phenomena—viz., between the cause, the mode of action of the cause of disease, and the attendant results.

In the study of Pathology, therefore, we have regard to—

I. Ætiology—the cause of disease.

II. Results of Disease:—

(1) *Pathological Anatomy*—

(*a*) Macroscopic—Naked-eye Anatomy.
(*b*) Microscopic—Minute Anatomy.

(2) *Pathological Chemistry*.

III. Processes of Disease.

I. **Ætiology** treats of the causes of disease. These are innumerable, but may be divided into—

(1) Predisposing Causes, viz., all such factors as render a person liable to contract disease—*e.g.*, age, sex, inherited tendency, occupation, mode of life, etc.

(2) Exciting Causes. — Those which are the more immediate producers of the disease. They are subdivided into—

(*a*) *Mechanical* — *e.g.*, external violence, internal obstruction of orifices, etc.

(*b*) *Chemical—e.g.*, poisons, whether taken in from without, as arsenic, etc., or found in the blood, due to defective excretion.

(*c*) *Vital*—those causes to which the various contagious and infectious diseases are most probably due—*e.g.*, parasites, etc.

II. Results of Disease—

(1) Pathological Anatomy and Histology teach the naked-eye and microscopic changes which result from the various diseased processes.

(2) Pathological Chemistry gives us an insight into the chemical composition and probable antecedents of morbid products of disease.

III. Processes of Disease.—The processes of disease are essentially modifications in the nutritive powers and properties of cell elements—these modifications being either an exaggeration or diminution of the normal processes of nutrition, and the consequent functional derangements.

Pathological processes, therefore, may be classed as follows:—

(1) Altered States of the Blood and Circulation.

(2) Altered States of Nutrition.

(3) Aplasia or Hypoplasia—by which is meant a defect in the development of cells giving rise to the various *Malformations*. This will not be treated of in this part of the work.

II. DIVISION.—For convenience of study, Pathology is often divided into—

I. **General Pathology** which treats of morbid processes apart from the individual tissues or organs in which they may occur.

II. **Special Pathology** which treats of these same processes and of their special characteristics, as they affect special tissues and organs. Thus, in General Pathology, we treat of inflammation without reference to any one tissue that it may affect. In Special Pathology, we treat of inflammation as it occurs in the lungs, kidneys, liver, etc. etc.

GENERAL PATHOLOGY.

GENERAL PATHOLOGY may be treated under the following headings:—

SECTION I.—**Altered Conditions of the Blood and Circulation.**

(1) Hæmorrhage.
(2) Hyperæmia—
Dropsy.
(3) Anæmia.
(4) Thrombosis.
(5) Embolism.
(6) Infarction.

SECTION II.—**Inflammation and its Results.**

SECTION III.—**Altered Conditions of Nutrition.**

1. RETROGRESSIVE.—Nutrition impaired or arrested.
   (1) Degenerations and Infiltrations.
   (2) Atrophy.
   (3) Necrosis.
   (4) Gangrene.
2. PROGRESSIVE.—Nutrition increased.
   (1) Repair of Wounds, etc.
   (2) Hypertrophy.
   (3) Tumours.

SECTION IV.—**Parasites and Parasitic Diseases.**

(1) Animal.
(2) Vegetable.

Section I.

ALTERED CONDITIONS OF THE CIRCULATION.

Altered Conditions of the Circulation may be discussed under the following headings:—

1. Hæmorrhage.
2. Hyperæmia—
   Dropsy.
3. Anæmia.
4. Thrombosis.
5. Embolism.
6. Infarction.

Section I.

ALTERED CONDITIONS OF THE CIRCULATION.

I.—HÆMORRHAGE.

I. **Definition**—Hæmorrhage may be defined as the escape of the blood with all its constituents from the blood-vessels—arteries, veins, and capillaries—either individually or collectively.

II. **Division**—Hæmorrhage may be divided—

1. According to its **Cause** into—
   - (*a*) Traumatic—due to wounds, etc.
   - (*b*) Spontaneous—due to disease.

2. According to its **Quantity** into—
   - (*a*) Petechiæ.—When the hæmorrhage occurs as small defined red or brown spots.
   - (*b*) Ecchymosis.—When the bleeding is more extensive.
   - (*c*) Parenchymatous.—When the blood escapes from all the vessels of a part at one and the same time.
   - (*d*) Hæmorrhagic Infarction.—Where the solid tissues are completely infiltrated with escaped blood.
   - (*e*) Hæmatoma.—Where the effused blood forms a tumour or cyst.

3. According to its SITUATION into—

(*a*) EPISTAXIS—Bleeding from the nose.

(*b*) HÆMATEMESIS—Bleeding from the stomach.

(*c*) HÆMOPTYSIS—Bleeding from the lungs.

(*d*) HÆMATURIA—Bleeding from the urinary organs.

(*e*) METRORRHAGIA—Bleeding from the uterus.

III. CAUSES—

1. Traumatic Hæmorrhage is due to wounds, cuts, etc. (For this, see Surgical Works.)

2. Spontaneous Hæmorrhage is due to disease, and occurs—

(1) per RHEXIN—Rupture.

(2) per DIAPEDESIN.

(1) Hæmorrhage per Rhexin.—Rupture is due—

(*a*) To disease of the vascular walls, as in atheroma, aneurism, etc.

(*b*) To sudden increase of blood pressure, though, in this case, the hæmorrhage never occurs without previous disease of the vessels.

(*c*) To blocking of the blood-vessels by emboli, causing changes in the walls of the vessels and subsequent rupture.

(*d*) To changes in the blood itself, as seen in certain diseases—*e.g.*, typhus, septicæmia, scurvy, etc., and in some forms of phosphorus poisoning.

(2) Hæmorrhage by Diapedesis.—In this case the blood escapes from the vessels (veins and capillaries) without actual rupture of their walls. It is doubtful, however, whether this process ever occurs, though it is said to be due to changes or to defective structure in the vascular walls.

Hæmophilia or Hæmorrhagic Diathesis is a constitutional tendency to bleed from slight causes. It is a hereditary condition. No change of structure has been found in the vascular walls.

IV. CHANGES IN THE EFFUSED BLOOD—

1. When fresh, the effused blood has the colour of arterial or venous blood according to its source; but after a time, the colour changes, becoming brown, blue, green, and yellow.

2. Part of the blood is absorbed and carried away by the lymphatics, and excreted by the urine.

3. The stroma of the red corpuscles disappears, and part of the hæmoglobin thus liberated is diffused into the surrounding tissues, there undergoing chemical change, and part is carried by white corpuscles to other situations.

(For further changes, see "Pigmentary Degenerations and Thrombosis.")

II.—HYPERÆMIA.

I. DEFINITION—Excess of blood in the tissues and organs of the body.

II. DIVISION—Hyperæmia may occur as:—

1. A General Condition—Plethora.—Where there is excess of blood as a whole. This probably only exists as a temporary condition—thus, after bloodless amputation of a limb, there is, for the time being, an excess of blood in the body.

2. A Local Condition.—Where there is excess of blood in a particular part or organ.

LOCAL HYPERÆMIA.

I. DEFINITION—This condition is one in which there is more or less increased fulness and redness of the affected parts, from their containing more than their normal amount of blood.

II. DIVISION—Local Hyperæmia may be divided into—

1. Arterial (ACTIVE — CONGESTIVE) — depending upon increased inflow of blood to a part.
2. Venous (MECHANICAL—PASSIVE)—due to decreased outflow from the veins of a part.

I.—ACTIVE HYPERÆMIA

(Arterial Hyperæmia—Acute Congestion).

I. DEFINITION—Active hyperæmia is chiefly a physiological process, and is defined as the congestion of a part due to increased inflow of arterial blood. As far as it is pathological, it depends on the following causes.

II. CAUSES—All the conditions determining an increased inflow of blood to affected parts; hence, we have—

1. Collateral Hyperæmia.—Where, owing to the constriction of the blood-vessels of a part, and the consequent diminution of blood supply thereto, there is an engorgement of other areas.

2. Idiopathic Hyperæmia.—A condition where there is dilatation of blood-vessels from—

(*a*) Paralysis of vaso-motor nerves—*neuro-paralytic hyperæmia.*

(*b*) Stimulation of the vaso-dilator nerves—*neuro-tonic hyperæmia.*

(*c*) Direct injury or weakening of the vessel walls by contusions, etc.

3. Reflex Hyperæmia.—Hyperæmia of one part on stimulation of another.

III. EFFECTS OF ACTIVE HYPERÆMIA—

(*a*) Accelerated blood flow, with dilatation of the vessels. This may be transient or permanent —the latter leading to stagnation and stasis. There may also be pulsation in the veins.

(*b*) A bright red colour of the affected part.

(*c*) A local rise of temperature, in superficial parts, of ½° to 3° C., but still the temperature is never above that of the internal organs.

(*d*) Slight or severe œdema and dropsy.

(*e*) Increased secretion, if it be the vessels of a gland that are affected.

(*f*) Hypertrophy of the tissues.

II.—PASSIVE HYPERÆMIA

(Venous or Mechanical).

I. DEFINITION—By passive hyperæmia we mean an excessive amount of blood in any part from obstruction to the venous outflow. Owing to the blue colour of the affected parts, it is often called Cyanosis.

II. CAUSES—These may be General or Local.

1. General Causes—

(*a*) Defective arterial pressure due to obstructive heart disease—*e.g.*, mitral stenosis (obstruction of the mitral orifice) causes general venous engorgement.

(*b*) Gravity, which acts by retarding the return of blood to the heart.

(*c*) Damaged innervation of the arterioles.

2. Local Causes—

(*a*) Pressure on veins from without by tumours, etc.

(*b*) Disease of the walls of the veins.

(*c*) Obstruction to flow in the veins from thrombi and emboli.

III. Effects of Venous Hyperæmia—These effects vary according as to whether the obstruction be—(*a*) *rapid and total*, or (*b*) *partial and long standing*.

1. Rapid and Total Obstruction causes engorgement, swelling, lymph transudation, lividity, coldness, and possibly necrosis of the part.

2. Partial and Long Standing Obstruction—

(1) On the Vessels.

(*a*) The *larger venules and veins* become elongated, tortuous, dilated, and their valves are rendered incompetent. Then follows chronic venous congestion and cyanotic induration. The inner coats of the vessels thicken and may become calcified.

(*b*) In the *smaller venules and capillaries* much similar results follow. They become varicose, dilated, and press upon surrounding parts. Their walls may thicken, or the vessels may rupture giving rise to hæmorrhage.

(2) On the Blood.

Sometimes the blood in the vessels tends to coagulate, the clot first becoming fatty and afterwards calcareous (vein stones).

(3) On the Surrounding Tissues.

(*a*) Pressure effects—viz., atrophy, etc.

(*b*) Pigmentation.

(*c*) Fatty degeneration from impaired nutrition.

(*d*) Fibrous overgrowth from increased blood supply.

(*e*) Dropsy and œdema.

DROPSY.

I. DEFINITION — Dropsy may be defined as an excessive accumulation of lymph in connective tissue spaces, and in the greater cavities of the body. Normally, all the tissues are bathed in lymph which transudes from the blood-vessels, and is again taken up by the lymphatics and veins and passed back into the venous circulation. Now, a transudation of lymph beyond the capacity of the lymphatics and the veins to remove it, constitutes Dropsy.

II. DIVISION—Dropsy may be divided into:—

1. General—Where the effusion of fluid takes place more or less generally throughout the body.

2. Local—Where particular tissues or organs are affected:—

(*a*) ANASARCA.—Dropsy of the integumentary structures.
(*b*) ŒDEMA.—Dropsy of the connective tissues and solid organs.
(*c*) ASCITES.—Dropsy of the peritoneal cavity.
(*d*) HYDROPERICARDIUM, HYDRONEPHROSIS, HYDROCEPHALUS, HYDROTHORAX, and similar terms, sufficiently indicate by their names the regions affected.

III. CAUSES OF DROPSY—

1. Of General Dropsy are—

(*a*) Obstructive Heart Disease—Cardiac Dropsy.
(*b*) Lung Disease—Pulmonary Dropsy.
(*c*) Kidney Disease—Renal Dropsy.
(*d*) Certain Cachectic states.

CARDIAC and PULMONARY DROPSY are purely mechanical in their production, being due to obstructed blood flow, accompanied by congestion. They manifest themselves in organs near the heart—*e.g.*, liver and lungs, and also in the more dependent parts—*e.g.*, legs, etc., owing to the action of gravity, and in serous cavities.

Renal Dropsy and Dropsy due to Cachexia are not so simple in their causation. They are in all probability due to changes in the vessel walls, caused by the watery state of the blood, and by some noxious substance circulating in the blood (Ziegler).

2. Local Dropsy may be due to—

(*a*) Direct obstruction to the local circulation, *e.g.*, venous obstruction.

(*b*) Obstruction in the lymphatics.

(*c*) Damage by inflammation or other like processes.

IV. Characters of the Affected Parts—

1. Tissues that are œdematous swell up, look pale, are doughy, pit on pressure, and if cut into, a clear watery fluid exudes.

2. Organs such as the kidney become enlarged, but do not contain so much fluid, though, when cut into, the surface of the section is moist and glistening.

3. Dropsical Cavities are enlarged, and contain a clear pale yellow fluid with an alkaline reaction.

The colour of the affected part varies according to the amount of blood therein.

V. Characters of Dropsical Fluid—

1. Dropsical fluid closely resembles dilute lymph. It is a clear colourless or light straw tinted liquid, containing very few cellular elements, these consisting mostly of oil globules, leucocytes, red blood corpuscles, and changed epithelial cells. The presence of blood or bile will change its colour.

2. It differs from lymph—

(*a*) In having less albumen.

(*b*) In being of a lower specific gravity.

(*c*) In not coagulating spontaneously.

(*d*) Sodium salts are in excess.

3. In kidney disease, it contains urea; in diabetes, it contains glucose.

VI. NATURE OF THE DROPSICAL PROCESS—
The escape of lymph from the blood-vessels may be due to—

1. EXCESSIVE TRANSUDATION OF LYMPH from—
   (1) Altered relations of *Pressure* in the vessels—
      (*a*) Diminished pressure outside.
      (*b*) Increased pressure inside.
   (2) Alterations in the *Capillary Walls.*
   (3) Alterations in the *Composition of the Blood.*

2. DEFECTIVE ABSORPTION OF LYMPH from—
   (1) Obstruction in the absorbents, whether veins or lymphatics.
   (2) Alterations in the vessel walls.

1. Excessive Transudation of Lymph—

I. The action of DIMINISHED EXTERNAL PRESSURE, as a cause of excessive transudation of lymph, is well seen in the action of dry cupping, and also in the circumstance that dropsy occurs more readily in those parts in which the elasticity is least marked—*e.g.*, eyelids, scrotum, etc. Moreover, increased external pressure leads to absorption of dropsical fluid—*e.g.*, in treatment of hydrocele.

INCREASED INTERNAL PRESSURE, leading to excessive transudation, may have its source—

1. In *obstruction to the outflow—i.e.*, venous obstruction; this being the great cause of lymph transudation, thus—

(*a*) General venous obstruction due to valvular disease of the heart causes dropsy of the legs, serous cavities, etc. Heart disease alone, however, will not cause dropsy, unless there be present some other condition favourable to its production.

(*b*) Obstruction of the portal circulation causes ascites.

(*c*) Obstruction to the veins of Galen causes chronic hydrocephalus.

2. In *increased inflow.*—This is a cause of dropsy only when there is extreme vaso-motor paralysis, and damage to the walls of the vessels.

II. Altered Conditions of the Walls of the Capillaries—

In all cases of dropsy there is some change in the vascular walls—especially in the lining endothelium—of such a nature as to render them more permeable. Thus, in inflammation and in waxy degeneration, there are changes in the vascular walls which increase the tendency to transudation of fluid through them.

III. Altered Conditions of the Blood—

1. If the blood be more *watery* than usual, as is the case in hydræmia, we get dropsy, though this condition of the blood *per se* is not enough to establish the dropsy, unless other factors are also present; for artificial hydræmia, produced by injecting water into the vessels of a dog, does not cause any excessive transudation of lymph.

2. The presence of excess of the *normal constituents* of the blood—*e.g.*, albumen, or of foreign substances—*e.g.*, arsenic, urea, etc., tends to produce dropsy.

3. Altered conditions of the *albuminous constituents* of the blood also cause dropsy, but, as yet, little is really known of the nature of the process in this case.

2. Defective Absorption of Lymph is the second great factor in the production of dropsy. It may be due—

(1) To obstruction in the lymphatics and veins.

(2) To altered conditions of the capillary walls.

Lymphatic obstruction alone rarely causes dropsy, owing to the plentiful collateral anastomosis. It may, however, increase a dropsy already established from any other cause. Thus, elephantiasis is a dropsy due to blocking of lymphatics by a parasite *(filaria sanguinis hominis).*

Altered conditions of the capillary walls result in defective power of absorption and consequent dropsy.

VII. EFFECTS OF THE NERVOUS SYSTEM—

1. Certain nerve lesions are thought to influence the production of dropsy — thus, in paralysis, œdema of the affected limbs sometimes takes place.

2. Again, dropsy may be due to paralysis of vaso-motor nerves, or to damaged nutrition and loss of elasticity in the tissues through injury to their nerves.

All the above-named conditions—viz., venous obstruction, weak heart, vaso-motor paralysis, altered states of the blood, damaged nutrition of the vascular walls, loss of elasticity, and action of gravity, do not act alone but collectively, and help each other in the production of dropsy.

VIII. SEQUELÆ—

1. Resolution—in which case the dropsical fluid is removed by the opening up of the collateral venous channels, and by increased absorption by the lymphatics.

2. Damage to the nutrition of the affected parts from pressure, etc.

III.—ANÆMIA.

I. DEFINITION—Anæmia is defined as a defect in the quantity or quality of the blood.

II. DIVISION—

I.—GENERAL ANÆMIA.

(Oligæmia.)

This is a deficiency of blood throughout the body.

Other Terms—

1. SPANÆMIA.—Poverty of the blood in normal constituents.

2. HYDRÆMIA.—Excess of water in the blood from defective elimination.

3. ANHYDRÆMIA.—The opposite condition in which the albuminous elements of the blood are normal, but the salts and water are deficient.

II.—LOCAL ANÆMIA.

(Mechanical—Ischæmia.)

I. DEFINITION—Local anæmia is the condition characterised by a deficiency of blood in individual tissues or organs from diminished arterial supply.

II. CAUSES—

1. Hæmorrhage.
2. Excessive drain of blood to other parts.
3. Narrowing or obstruction of vascular channels—
   (*a*) By disease—*e.g.*, atheroma.
   (*b*) By external pressure from new growths, etc.
   (*c*) By ligature.
   (*d*) By thrombi and emboli.
4. Excessive contraction of arterioles—
   (*a*) From paralysis of vaso-dilator or stimulation of vaso-constrictor nerves, as seen in long-continued applications of cold or use of drugs such as opium, or in certain diseases—*e.g.*, Raynaud's disease.
   (*b*) From direct stimulation of the muscular walls by poisons circulating in the blood.

III. CHARACTERS OF THE AFFECTED PARTS—Parts that are anæmic become paler and colder than usual, are less tense and less firm, and on post-mortem examination show a deficiency in the normal amount of blood.

IV. SEQUELÆ—

1. Impaired nutrition and function, followed by—
   (*a*) Fatty degeneration.
   (*b*) Atrophy.
   (*c*) Necrosis.

2. Rise of blood pressure, and dilatation of the blood-vessels of other parts.

(Other changes, see "Thrombosis" and "Embolism.")

IV.—THROMBOSIS.

I. DEFINITION — Thrombosis is the intra-vascular clotting of the blood during life, the coagulum being called a THROMBUS, in contra-distinction to a CLOT —a coagulum formed after death.

II. CAUSES—

1. Disease or injury to the walls of the blood-vessels—*e.g.*, section or rupture of the vessels; irregularities in their walls; foreign bodies in the vessels.

2. Retardation of the circulation from weak heart; from dilatation or peculiarities of arrangement of the vessels—*e.g.*, vascular plexuses—from the action of gravity; from external pressure, etc.

3. Abnormal states of the blood, as seen in septic fevers, gouty conditions, puerperal state, hyperinosis.

III. SITES—Thrombi may occur:—

1. IN THE HEART—
   (*a*) In the right auricular appendix.
   (*b*) In the right ventricle—the clot in this case often being continuous with one in the auricle, and extending into the pulmonary artery.
   (*c*) In the left auricular appendix, as seen in mitral stenosis, the thrombus starting in the appendix and filling up a great part of the auricle.
   (*d*) In valvular diseases of the heart, the vegetations on the aortic and mitral valves are mostly thrombi.
   (*e*) In the apex of the left ventricle when dilated by disease — in the saccules between the columnæ carneæ.
2. IN ARTERIES—
   (*a*) From roughening of their walls, as in atheroma.
   (*b*) From narrowing of the vascular channels.
   (*c*) From injury to the vascular walls, as in aneurisms.

3. In Capillaries.—Thrombosis is rare, but it may occur as a result of inflammation or of local damage.

4. In Veins.—This is the most common situation in which thrombi occur, owing to obstructed outflow from dilatation of the veins.

IV. Varieties of Thrombi—

1. Obliterating (Obstructing—Total).—Where the thrombus fills the vessel.

2. Parietal.—When it forms on some part of the vessel wall.

3. Peripheral (Channelled).—When it coats the inside of the vessel, leaving a passage through its centre.

4. Free thrombi.

5. Saddle-shaped.—When the coagulum becomes impacted upon the bifurcation of a blood-vessel.

V. Characters of Thrombi—Thrombi vary in appearance according as they are formed in stagnated blood, or in blood that is still circulating, and are known as Red and White thrombi respectively.

1. **Red Thrombi** originate in stagnated blood, which consequently coagulates "*en masse*." Such thrombi are seen after the ligature of an artery.

Macroscopic Structure.—At first the thrombus appears like a mass of freshly-drawn coagulated blood, and has a dark red or brown colour, is soft, uniform on section, and adheres to the wall of the vessel. After a time it shrinks, becomes firmer, drier, less elastic, and paler, though it still retains its red colour. In this stage the thrombus is usually found.

Microscopic examination shows that a red thrombus is composed of red corpuscles densely packed together, with but few fibrin filaments intervening. Sometimes the corpuscles appear as though merely fused together.

2. White Thrombi are formed while the blood is still circulating.

MACROSCOPIC STRUCTURE.—White thrombi have a grey white, pale, or mottled appearance, are striated, stringy, and laminated. They are firm, not easily broken down, and are closely adherent to the walls of the vessels.

MICROSCOPIC STRUCTURE.—White thrombi consist of homogeneous or finely granular masses of fibrin—the so-called "granular fibrin"—really a mass of disintegrated blood plates. The outer part of the thrombus, however, is composed of blood plates in an unchanged condition, and of fine filaments of fibrin entangling white corpuscles and a few red corpuscles in its meshes.

Post-mortem Clots, as distinct from thrombi, are characterised by their soft, black-red, gory, gelatinous, watery appearance. They are never adherent to the walls of the vessels, but can be drawn out of them as long strings.

Ante-mortem Clots, on the other hand, are usually opaque, firm, dry, striated, less elastic, and are adherent to the vascular walls.

VI. INTIMATE PATHOLOGY AND MODE OF FORMATION OF THROMBI—Thrombosis is usually said to depend on two conditions, viz.—(1) on changes in the walls of the blood-vessels, and (2) on retardation or stoppage of the blood current (Virchow); but neither of these factors alone is the sole agent in the production of thrombosis, for (*a*) it has been shown that intact vascular walls are not essential to the fluidity of the blood, and (*b*) that the stoppage or slowing of the blood stream only indirectly favours coagulation.

Again, in the production of artificial thrombi, it has been stated that the leucocytes, which gather on the walls of the blood-vessels, break down, and, in so doing, liberate a

ferment which, by its action, brings about a local formation of fibrin. But there is now evidence that no such disintegration of the white corpuscles takes place, but that the leucocytes migrate through the vessel walls. Moreover, leucocytes can be found intact in freshly formed blood clot.

Blood Plates (Blut plättchen).—In normal blood, and in recently drawn blood, prevented by any means from coagulation, there can be seen small circular or oval bodies called *blood plaques* or *blood plates*, which measure from 1·5 to 3·5 micro-mill. in diameter. They are non-nucleated, but have a distinct outline, and consist of a homogeneous or finely granular protoplasm. In number they average from 2 to 300,000 in a cubic mill. of blood, and are in the proportion of 1 to 18 or 20 of the red blood discs. They are increased in number in consumption, in typhoid fever, and in pernicious anæmia.

Reactions.—Blood plates stain easily with methyl violet and with logwood, slightly with methyl blue, but not with carmine When brought in contact with foreign substances, or when the blood has been drawn for sometime from the vessels, the blood plaques change their shape, become jagged at their edges, viscous, and tend to cling together and to form granular masses.

These blood plates play an important part in the formation of thrombi, especially of white thrombi, for—

(*a*) When there is any damage to the walls of the blood-vessels the blood plates adhere to the injured spot, and there form small projections upon which leucocytes and fibrin are soon deposited. Layer after layer is thus formed, each successive lamina commencing by a deposit of blood plates, followed by a deposition of fibrin, giving rise to a laminated clot.

(*b*) Under normal circumstances the blood plates circulate in the axial stream, but when the blood current is

retarded the plaques and the leucocytes fall out of the axial into the peripheral stream. If the circulation becomes still slower the leucocytes return to the axial stream, but the blood plates gather more and more upon the walls. If the circulation be entirely arrested all the corpuscles are mingled together indiscriminately, but the blood plates cling to the walls and lay the foundation of the future thrombus.

VII. Mode of Increase of Thrombi—

Thrombi may grow rapidly, or, as is more commonly the case, slowly by accretion. This latter especially occurs in arteries, where the rapid and vigorous circulation hinders their increase.

Thrombi, once formed, generally extend in the direction of the heart, though they may form also in the opposite direction. They often start in the pouch of one of the veins, and extend thence to the next collateral branch. In some cases they reach a great size, and have been found extending from the foot to the inferior vena cava.

VIII. Changes in Thrombi—

1. Resolution.—Thrombi are sometimes removed, but by what process is not yet understood.

2. Contraction.—Thrombi often shrink and become adherent to the walls of the vessels.

3. Discoloration.—In red thrombi the red blood corpuscles break down and their stroma disappears. The hæmoglobin thus set free is partly taken up and removed by leucocytes, and partly deposited as hæmatoidin crystals within the thrombus. Thus, the thrombus loses its original dark colour.

4. Fatty Degeneration.—Where the coagulum changes into a yellow granular mass—the blood plates resisting the change the longest.

5. Calcification.—Giving rise to the so-called vein stones—phleboliths.

6. Softening—

(*a*) *Simple or Red Softening.*—Where the thrombus becomes more or less fluid, and of a red or white colour according to its original nature. The fluid mass consists of debris of corpuscles, fibrin, fat, and pigment granules.

(*b*) *Infective or Yellow Softening.*—In which case, besides the above-named changes, we find septic poisoning and the presence of micrococci.

7. Organisation.—By which we mean the replacement of the fibrin and corpuscles by vascularised fibrous tissue. (See Repair—Ligature of Vessels.)

IX. Results of Thrombosis—

1. Changes in the Walls of the Vessels.—These first of all become thickened and infiltrated with leucocytes, and then, like the thrombus itself, shrink and atrophy, leaving a fibrous cord. If the thrombus undergoes septic softening, a process of acute inflammation is set up, and the endothelial lining of the vessels dies and is cast off.

2. Obstruction to the Circulation.—The consequences of which vary—

(*a*) According to the size and position of the occluded vessel.

(*b*) According to the nature of the tissue affected.

(*c*) According to the rapidity of the onset of the obstruction.

(See "Dropsy," "Anæmia," etc.)

3. Embolism.—Where part of the thrombus becomes detached, and is carried by the blood stream to some vessel too small to allow it to pass.

V.—EMBOLISM.

I. DEFINITION—Closely allied to thrombosis are the phenomena of embolism, which may be defined as the plugging of vessels by the impaction in them of clots or of foreign bodies circulating in the blood; the matter thus impacted being called an EMBOLUS.

II. VARIOUS KINDS OF EMBOLI—

1. Small portions of thrombi that have become detached and been swept away into the circulation. This is the most common form of embolus.

2. Heart vegetations, atheromatous masses.

3. Parts of tumours which have grown into the blood-vessels.

4. Parasites, air, fat, gas, pigment granules, or any foreign body which may have found its way into the blood current.

III. DIVISION—Emboli are divided into :—

1. SIMPLE.
2. INFECTIVE or SEPTIC.

IV. SITES—Emboli occur :—

1. In the systemic and pulmonary arteries, and also in capillaries.

2. In the portal vein, owing to its branching like an artery.

Emboli are usually carried along in the direction of the blood current, till they reach the bifurcation of a vessel too small to allow of their passage. Here they become arrested and form a plug in either or both branches, and upon this plug successive layers of clot are deposited until the vessel becomes occluded, and a thrombus is formed on both sides of the block.

V. EFFECTS OF EMBOLI—

1. Simple Emboli.—The effects of simple Emboli are purely mechanical, and are such as result from stoppage or obstruction of the circulation. They vary, however, according as the affected vessels are—

(1) Non-terminal arteries.
(2) Terminal arteries.

(1) When the occluded vessel is a NON-TERMINAL ARTERY—*i.e.*, one in which there is collateral anastomosis, then—

(*a*) If the circulation be soon re-established through the side branches, there will be little or no bad effects. The circumstances interfering with this re-establishment are—

1. A weak heart.
2. Weakness of the vascular walls and degeneration.
3. Double or multiple obstruction.

(*b*) If the circulation is not re-established, there results—

1. Anæmia and consequent nutritive changes, fatty degeneration, anæmic necrosis.
2. Hæmorrhage, from stoppage of the normal channels of flow.
3. Dilatation of the vessels and aneurism—*e.g.*, in the brain.
4. Gangrene, if it be external parts that are affected.

(2) When the occluded vessel is a TERMINAL ARTERY—one in which there is no collateral anastomosis, then we get the phenomena of EMBOLIC INFARCTION. (See page 23.)

2. Septic Emboli.—These give rise to new growths, or to inflammation at the point where the obstruction occurs. (See "Embolic Abscess.")

VI. Changes in Emboli—

1. An embolus may be absorbed (rare).
2. It may organise like a thrombus.
3. It may soften, shrink, calcify.
4. It may become septic.

VII. Other Forms of Embolism—

1. Secondary Emboli (recurrent).—Where parts of a secondary thrombus become detached, and are carried to the heart, and then lodged in some other part of the circulatory system.

2. Miliary Emboli.—Where a number of minute fragments of thrombi become detached in great numbers and block up several vessels. This is especially seen in cases of ulcerative endocarditis, where the afferent arterioles of the kidney may become occluded by minute emboli.

3. Oil Emboli—

(1) In fractures of bone, globules of oil sometimes pass into the veins and are carried to the heart, and become lodged in the blood-vessels of the lungs, etc.

(2) In diabetes, oil gathers in the blood *(lipæmia)*, and passes thence to the pulmonary capillaries and to the kidneys, etc.

(3) In glanders, and in some cancers.

VI.—INFARCTION.

I. Definition—Closely connected with Embolism is the phenomenon of Infarction, which may be defined as the result which follows the blocking of a *terminal* artery by Emboli, etc.—the solid wedge-shaped mass thus formed being called an Infarct.

II. TERMINAL ARTERIES are those in which there is no collateral anastomosis. They occur—

1. In the retina.
2. In the cortex of the brain, and in basal ganglia.
3. In the cortex of the kidney.
4. In the spleen.
5. On the surface of the liver, lungs, and heart.

Infarction may occur in relation to any of the arteries in the above-named situations, and also in the portal vein, from its branching like an artery.

III. CHARACTERS OF THE AFFECTED PART—Infarcts vary somewhat in character according to the situation in which they occur, thus—

1. In the **Spleen** infarcts appear, at first, either as pale yellow or dark red patches, which vary much in size, from a pin-head to one half the size of the entire organ. They are wedge-shaped on section—thus corresponding in form to the area of the occluded vessel,—their base is raised above the surface, and their apex is rounded and directed inwards. Under the microscope the blood-vessels of the spleen are seen to be dilated, and the splenic pulp, veins, and capillaries, are engorged with blood. After a time the central part of the Infarcts becomes paler in colour, but there remains a marginal zone of congestion, an appearance that is especially characteristic. Soon, however, necrotic changes set in, due to malnutrition, and the infarcted area becomes transformed into a granular mass of fat and debris. The connective tissue at the margin of the infarcts begins to proliferate and forms a distinct fibrous capsule round the affected area. This now shrinks, leaving a depressed stellate cicatrix.

2. In the **Kidney,** somewhat similar dark red or mottled grey wedge-shaped masses are met with. They vary much in size, their base is directed to the surface of the

organ, their apex inwards, and they are surrounded by the characteristic marginal zone of congestion. Ultimately they necrose and become encapsuled and shrink. If the kidney tissue should necrose before the engorgement can take place, the so-called "white wedges" are formed.

3. In the **Lungs** infarcts may be from half-an-inch to two inches in diameter. They are wedge-shaped, rusty-red masses, situated on the free border or surfaces of the lungs. Their exact mode of formation is as yet by no means clear.

IV. RESULTS OF INFARCTS may be classed under two heads:—

(1) Vascular Changes.
(2) Tissue Changes.

1. Vascular Changes. — When a terminal artery becomes blocked—*e.g.*, by an embolus—the area supplied by the occluded vessel should naturally be anæmic from loss of its blood, but such is not the case, for the part soon becomes engorged with blood. Two explanations have been given of this phenomenon:—

(1) In the first place, the engorgement is said to be due to Reflux through the veins — Venous Reflux (Cohnheim).
(2) In the second place, to Collateral Capillary Anastomoses (Litten).

1. REFLUX THEORY.—When a terminal artery is blocked the arterioles of the affected area contract and drive on the blood into the veins, and as a consequence there is a great fall of blood pressure. The venous pressure being in excess of this, blood regurgitates through the veins into the capillaries and arterioles of the occluded region, thus giving rise to engorgement. It is also stated that diapedesis occurs at the margin of the infarcts, owing to loss of nutrition in the vascular walls.

Experiment—

Cohnheim injected small pellets of wax into the aorta of the frog, and of these some were carried by the blood stream to the arteries of the tongue, the lateral branches of which are terminal vessels. As a consequence, infarction took place; and Cohnheim states that he observed regurgitation from the veins into the affected area, resulting in engorgement, stasis, and diapedesis.

2. Collateral Anastomoses.—Litten, on the other hand, maintains that the engorgement is due to the blood reaching the infarcted area through the minute arterial capillaries which anastomose with those of the occluded vessel.

Experiments—

(1) If the renal artery be tied, an infarct is formed in the kidney. Now, this engorgement is not due to reflux through the vein, for it still occurs even though the blood be allowed to escape through the cut end of the vein, or if the vein be at the same time ligatured. Again, the engorgement is seen to commence underneath the capsule of the kidney, and round the base of the Malpighian pyramids, where the fibrous tissue of the calices is attached. Now, these circumstances point to the fact, that the engorgement has its origin in the capillary anastomoses which occur in these sites, between the vessels of the kidney on the one hand, and those of the capsule and ureters on the other.

(2) Finally, if the renal artery be rendered a truly terminal artery by stripping off the capsule and ligaturing the ureters, then, if you ligature the renal artery, no infarction follows, even though the vein be not tied. But if you now ligature the vena cava above the origin of the renal vein, and thus increase the venous pressure, an infarct is the result.

2. Tissue Changes.—The tissue changes which result from the blocking of a terminal artery are of the nature of a *necrosis*. Some tissues resist these changes better than others: thus fibrous tissues are the most resistant, ganglion cells the least so.

Experiments—

(1) If the main artery to the ear of a rabbit be ligatured at its root, and the ligature be retained for eight or ten hours and then removed, we find that the blood-vessels of the ear become much dilated and congested, and œdema is the result—lymph, leucocytes passing through the vascular walls and infiltrating the surrounding tissues. If the ligature be allowed to remain for twenty-four hours, small extravasations of red as well as white corpuscles will be observed; and if the ligature remains *in situ* for forty-eight hours, the epithelium of the part will be cast off and the tissues will begin to necrose.

(2) Next, suppose a ligature be applied to the base of a frog's tongue, similar changes will be observed; the muscular fibres degenerate and perish.

(3) Again, if the renal artery be tied, and the kidney thus deprived of blood for two hours, the renal epithelium becomes detached from the tubules and is transformed into homogeneous granular masses. If the ligature be retained for six or eight hours, the connective tissue elements then begin to perish—they swell up, become fatty, caseous, and then calcify, and a complete destruction of the renal tissue follows.

These several phenomena explain the changes which occur in an infarct—for the blood filling the infarcted area coagulates, then changes colour, becoming brown and yellow. The central part of the infarct breaks down into a pulpy mass like pus (necrosis) and then dries up—the fibrous

tissue at the margin of the infarct being increased in amount, forms a definite capsule, and the whole mass shrinks, leaving the depressed stellate scar above referred to.

Infarcts of the Lung differ somewhat from those in the Kidney. They are especially met with in cases of mitral disease, with engorgement of the pulmonary circulation, and of the right side of the heart. Their exact mode of formation is doubtful—for (1) they may be due to embolism of the pulmonary veins, or (2) to hæmorrhage into the bronchi; for the blood-vessels of the bronchial walls may be much engorged and rupture, the blood being drawn into the bronchi, thus forming a wedge-shaped mass similar to an infarct of a vessel. Whether infarction of the Lung is due to blocking of the pulmonary artery is, to say the least, doubtful —for, except where the animal has been much weakened by previous loss of blood, it has been found impossible to produce an infarct by blocking of the pulmonary artery.

Section II.

INFLAMMATION AND ITS RESULTS.

1. Definition of Inflammation.
2. Sites of Inflammation.
3. Causes of Inflammation.
4. Signs of Inflammation.
5. Theory and Nature of the Process.
6. Phenomena of Inflammation—
    (1) In Vascular Structures.
    (2) In Non-Vascular Structures.
7. Varieties of Inflammation.
8. Results of Inflammation.

Section II.

INFLAMMATION.

I. Definition—

Inflammation may be defined "as the succession of changes which takes place in living tissues as a result of an injury, provided that the injury be insufficient to at once destroy their vitality" (Burdon Sanderson); or again, it may be defined "as the series of changes which take place as a consequence of the reaction of blood-vessels and tissues against the irritation inflicted upon them by some deleterious agent."

II. Sites—

Inflammation may affect almost any tissue or organ which possesses blood-vessels, or is in immediate relation to blood-vessels. It may therefore affect all tissues except a few epidermic structures, such as hair, etc.

III. Causes—

The causes of inflammation are manifold. They are called Noxæ; and are all such agents as can cause damage to blood-vessels and tissues.

They may be classed as—

1. Mechanical—*e.g.*, Pressure, etc.
2. Chemical.
3. Thermal.
4. Vital—*e.g.*, certain Organisms.
5. Poisons in the Blood.

The mode of action of Noxæ is—

(1) Directly on the vascular walls, and through them upon the tissues.

(2) On both blood-vessels and tissues simultaneously.

(3) On the tissues, and through them upon the vessels.

IV. SIGNS OF INFLAMMATION—

The local or clinical signs of inflammation are rubor, calor, tumor, dolor—redness, heat, swelling, pain.

The *redness* is due to dilatation of the blood-vessels, especially of the smaller veins and capillaries.

The *heat* is caused by the increased influx of blood into the inflamed part, not to any heat generated in the inflamed area itself.

The *swelling* is in part due to the excess of blood in the region, in part to the increased transudation of lymph from the blood-vessels, and in part to the loss of elasticity in the surrounding tissues.

The *pain* is probably caused by pressure, or by tension, or by the action of some chemical agent on the sensory nerves of the parts.

The above named phenomena, though sufficiently indicative of the presence of inflammation, are by no means the essential factors or features in the process, for one or more may at any time be absent and yet the essential characters of inflammation may exist.

V. THEORY OF INFLAMMATION may be stated as follows:—

1. That inflammation is a lowering of the vitality of tissue elements, not an increase in their nutritive activity.

2. That one essential factor in the production of inflammation is spreading damage to the walls of the blood-vessels, in consequence of which we have slowing of the blood stream, and escape of lymph and leucocytes.

3. That the small-celled infiltration, formerly regarded as having its source in the proliferation of tissue elements, is due to the escape of leucocytes from the vessels—the tissue elements themselves remaining passive, or else undergoing merely degenerative changes.

VI. PHENOMENA OF INFLAMMATION—

1. Vascular Changes.
2. Textural Changes.

I. Vascular Changes of Inflammation may be arranged as follows:—

(1) Active Hyperæmia.
(2) Slowing of the blood stream, and stasis.
(3) Lymph transudation.
(4) Leucocytes emigration, and escape of red corpuscles.
(5) Spread of Inflammation to surrounding parts.
(6) Resolution.

These several phenomena, though for convenience thus separately enumerated, take place simultaneously. They can be best observed in such transparent membranes as the mesentery of the frog. The process is essentially the same in warm-blooded and in cold-blooded animals.

1. ACTIVE HYPERÆMIA.—When the mesentery of the frog is irritated, *e.g.*, by exposure to the air, after a transient and unimportant stage of contraction of the blood-vessels, there immediately follows great dilatation, with a quickened rate of blood-flow. This is first observed in the arterioles, then in the capillaries and small veins. The arterioles then contract, but the veins and capillaries remain dilated.

2. SLOWING OF THE BLOOD STREAM.—After the initial hyperæmia and increased rate of blood-flow, the current becomes much slower, oscillating at first to and fro, and finally in the small veins and capillaries it comes to a standstill—STASIS.

3. Lymph Transudation.—The escape of lymph, which commenced in the stage of hyperæmia, ultimately becomes very pronounced, and causes local dropsy and œdema.

4. Leucocyte Emigration — Diapedesis. — Concomitant with the slowing of the blood current, the white corpuscles, which in the normal circulation occupy the axial stream, begin to fall out of the central stream and to gather on the walls of the veins, and to some extent also on the walls of the capillaries. Then by a process already familiar to you in your physiological studies, under the term diapedesis, the white corpuscles gradually pass through the vascular walls and infiltrate the surrounding tissues, thus giving rise to the small-celled infiltration so characteristic of inflammation. Red corpuscles also pass out of the blood capillaries in small jets, but without actual rupture of the vascular walls, and give rise to punctiform hæmorrhages often visible to the naked eye.

5. Spread of Inflammation.—If the original damage has been great, coagulation and thrombosis of the affected vessels is the result, the vitality of the part is lost, and the inflammation extends, by spreading damage to the vessels and to the surrounding structures.

6. Resolution.—Should the original irritant be removed or the cause which set up the inflammation cease to act, then, if the part has not been killed, the blood stream quickens and soon returns to its normal rate; the white corpuscles pass back into the circulation; the transuded lymph is re-absorbed, and the blood-vessels and tissues regain their normal appearance.

Detailed Discussion and Explanation of the above Vascular Phemomena:—

1. Active Hyperæmia, though a constant accompaniment of inflammation, is not essential to the process, for the hyperæmia may subside without any subsequent

inflammation, and we may have all the evidences of inflammation without previous hyperæmia.

Experiments—

(1) If caustic be applied to the membrana nictitans of the frog, it may set up inflammation without any hyperæmia.

(2) If mercuric nitrate be applied to the ear of the rabbit, it causes inflammation but no hyperæmia.

(3) When the rabbit's ear is exposed to heat or cold, we may get hyperæmia without any subsequent inflammation.

(4) If a small central spot of the frog's tongue be irritated there follows active hyperæmia of the affected area, with slowing of the blood stream, a certain degree of stasis, and gathering of the leucocytes in the peripheral stream. If there be no further damage, there is no lymph transudation and no emigration of leucocytes. In this case the probable cause of the active hyperæmia is the local action of the irritant on the ganglia in the walls of the vessels, and not damage to the vascular walls; for Cohnheim cut through all the structures of the tongue except the main artery and vein, and still produced hyperæmia when an irritant was applied in the region of the vessels. The vascular dilatation is also independent of the central nervous system, for it occurs after removal of the brain.

(5) If a still greater amount of irritation be applied to the tongue, sufficient to materially damage the vascular walls, then we find the following zones—(*a*) At the spot injured the blood flow becomes arrested, the blood in the capillaries coagulates, and there is a local death of the tissues. (*b*) Immediately outside the injured spot the vessels are permanently dilated, especially the veins, and there is

slowing of the blood stream and stasis; lymph exudation; leucocyte emigration. (*c*) Beyond this area there is a zone of dilated vessels, retarded blood flow, and partial stasis. (*d*) Still further from the central spot of irritation, the blood-vessels are dilated with the increased rate of blood flow. (*e*) Beyond this is the normal tissue and normal circulation.

2. Slowing of the Blood Stream. — This, the second phenomena of inflammation, is especially well seen in the veins and capillaries, and gives rise to a great accumulation of blood in the affected part. The cause of the slowing is not understood, but may be due, either—

(1) To some molecular change in the vascular walls; or

(2) To some change in the tissues outside the vessels.

3. Lymph Transudation commences in the first stage of the inflammation, and continues during its whole course.

1. The CAUSES of it are:—

(1) Increased permeability of the vascular walls.

(2) Increased intra-vascular pressure.

(3) Diminished elasticity in the surrounding tissues, and hence the amount of transudation varies according to the tissue in which it occurs, being greater and more quickly established on free surfaces and in subcutaneous tissues than in the more solid organs.

2. CHARACTERS OF THE TRANSUDED LYMPH.—It closely resembles dropsical fluid, but—

(1) Contains more albumen.

(2) More solid constituents.

(3) Coagulates better.

4. Leucocyte Emigration—Diapedesis—may be considered under three headings:—

1. Accumulation of Leucocytes in the Peripheral Stream.—This is a purely mechanical phenomenon, and can be at any time induced by slowing of the blood stream. It is said to be due to a change in the relative specific gravity of the corpuscles and of the blood plasma, but this is doubtful. It is certainly not due to the adhesiveness of the corpuscles to the walls of the blood-vessels.

2. Passage of Leucocytes through the Vascular Walls—

(1) This is a gradual process and does not take place all at once, but first a small part of the corpuscle passes through the vascular walls, and then the rest follows. The time occupied in the passage varies from eight minutes to half-an-hour or two hours.

(2) The passage is accomplished by the active movement of the corpuscles, for it does not occur when the corpuscles are dead.

(3) The presence of oxygen is essential to its accomplishment, for want of oxygen arrests the movement, whereas an increased supply of oxygen accelerates it.

(4) The damaged vascular walls are more permeable than normal, and this helps the passage of the leucocytes.

(5) Increased blood pressure may aid the emigration, but this is very doubtful, as some hold that the blood pressure in an inflamed area is decreased.

3. Function of the Emigrated Corpuscles—

(1) They aid coagulation.

(2) They remove dead products.

(3) They help in the formation of new tissue.

(4) They form pus corpuscles.

(5) Some return to the circulation, others settle down in the tissues.

5. Resolution.—This is brought about by cessation of the action of the irritants, and by repair of the vascular walls due to the *vis medicatrix* of the blood—the inflammatory process subsiding, and the circulation returning to the normal.

II. Textural Changes in Inflammation.—Having so far examined the vascular phenomena of inflammation, we shall next consider the changes in the Tissue Elements as distinct from those in the blood-vessels. They can be best studied in such tissues as Cartilage and the Cornea, for they are non-vascular.

The nature of these textural changes is, according to one view, a reaction of the tissues against the injury inflicted upon them, *i.e.*, reparative; according to another view, these tissue changes are associated with depressed vitality and degeneration, never with multiplication, of tissue elements, and increased nutritive activity, as stated by Virchow.

It is difficult to decide between these two theories, for, when we examine, say, the endothelium cells of an inflamed area, we find that they are swollen and detached, and that their nuclei have proliferated, while many of the cells have broken down and perished.

The same changes, *ceteris paribus*, may be seen in connective tissue cells, for, in the sites of the connective tissue corpuscles, there are found groups of cells, like white corpuscles, which are called *plasmodia*—plasma cells; and the question arises as to the origin of these cells or groups of cells, viz.:—

(1) Whether they are due to a degeneration and proliferation of connective tissue corpuscles of the affected part, or

(2) Whether they are leucocytes which have emigrated from the marginal vessels of the inflamed area, and have then fused together.

The latter is the more probable hypothesis.

Experiments:—

1. On Cartilage—

If a piece of cartilage be irritated without causing damage to the vessels at its margin, we find that the cell spaces of the cartilage matrix become enlarged, and contain proliferated cartilage corpuscles. Then the cartilage matrix becomes softened, stains more deeply with reagents, and finally undergoes solution, leaving large spaces containing many cells—*plasmodia*. This experiment, however, does not decide the origin of these clumps of cells, for the vessels at the margin of the cartilage are seen to penetrate the cartilage matrix and to ramify therein, and it is more than probable that the plasmodia are white corpuscles which have passed from these marginal vessels into the cell spaces, and have there fused together.

2. On the Cornea—

(1) If the cornea be injured, two sets of changes will be observed—(*a*) one at the seat of injury, and (*b*) the other at the periphery of the cornea.

(*a*) If a small central spot be injured by a slight scratch, then at the spot injured the part becomes opaque, the opacity lasting for two or three days and then subsiding, the cornea regaining its normal appearance.

(*b*) If through the central opaque spot a seton be passed, thus setting up more intense irritation, it will be found that, at the spot injured, the corneal corpuscles have become irregular in shape, and are showing signs of degeneration; and in the sites of the corpuscles—lacunæ and canaliculi—there are many small cells like leucocytes, though somewhat flatter, and also large multi-nucleated amœboid masses—*plasmodia*. At the *margin* of the cornea the blood-vessels are much dilated

and surrounded by leucocytes, and the conjunctiva is swollen, due to reflex irritation. Now, Cohnheim has shown that it is from these engorged vessels that the leucocytes have emigrated into the lacunæ and canaliculi.

(2) Again, by the use of zinc chloride solution, Senftleben killed the corneal corpuscles in a small localised area without directly affecting the marginal vessels, and under these conditions he found that the central spot remained clear, and that the corneal corpuscles underwent no change until the process of repair commenced, when they shot out processes and began to form new corneal tissue. If, however, the irritant be applied to the periphery of the cornea, and be of such intensity as to injure or destroy the anterior laminæ of the cornea, then the marginal vessels became dilated, the part became cloudy, due to infiltration of the tissues with emigrated leucocytes, and in the situation of the corneal corpuscles were found groups of cells—plasmodia.

Conclusion.—Thus we see that the small-celled infiltration, so characteristic of inflammation, has its source, in all probability, not in the proliferation of tissue elements, but in the migration of leucocytes from damaged vessels, and that the connective tissue elements remain passive until the commencement of the process of repair. (See "Repair.")

Moreover, from these several experiments on vascular and non-vascular tissues, we conclude that the essential element in inflammation is, not the initial hyperæmia, but damage to the vascular walls — the damage not being demonstrable by the microscope, but being a molecular change, probably of the nature of cloudy swelling of the endothelial cells, with a loosening of the cells, due to a giving way of the intercellular substance. This leads to an increased permeability of the vascular walls.

VII. VARIETIES OF INFLAMMATION. — Inflammation may be classed—

1. According to its **Duration**, into—

(1) ACUTE — in which case the inflammatory process commences more or less suddenly, is violent in type, and quickly subsides.

(2) CHRONIC—which may commence in a similar manner to the acute form, but usually comes on more slowly, and continues for a longer time. It exhibits all the phenomena of the acute form, but the blood-vessels remain dilated, and there is a continuous exudation of leucocytes.

2. According to the **Tissue affected**, into—

(1) PARENCHYMATOUS.—That which attacks the proper substance — the essential elements — the parenchyma — of an organ, as distinct from the mere supporting structures. It is at first a degenerative and destructive process, and when it affects glands, is closely allied to cloudy swelling; when it affects muscle, to coagulative necrosis.

(2) INTERSTITIAL—When the inflammation attacks the supporting structures—the framework of an organ—the connective tissue elements, as distinct from the parenchyma. If the process be acute, there is a tendency to suppuration; if the process be chronic, there ensues an overgrowth of the connective tissue—*cirrhosis*.

3. According to the nature of the **Exudation**, into—

(1) DRY, PLASTIC, FIBRINOUS.—When the exudation on the inflamed surface is small in amount and coagulates.

(2) CROUPOUS. — Often used synonymously with the above, but is especially applied to indicate the formation, on the inflamed surface, of a tough white or yellow covering called a *false membrane*.

(3) SEROUS.—When the exudation is very watery, and very similar to that which transudes from vessels that are engorged. It differs from it, however, in containing more

albumen and more leucocytes. If the fluid becomes turbid it is called SERO-PURULENT inflammation.

(4) CATARRHAL. — Which especially affects mucous surfaces, and surfaces covered by epithelium. It is characterised by an increased secretion, a mucous degeneration of the epithelial cells, and a swelling of the subjacent basement membrane. The exudation usually contains no fibrin and does not tend to coagulate, due probably to the fact that the epithelial cells have the power of preventing the coagulation.

(5) PURULENT, SUPPURATIVE, PHLEGMONOUS. — These terms are applied when the inflammatory exudation contains Pus, *i.e.*, a milky white coloured fluid, containing many leucocytes, and cells with usually one to three or more nuclei—Pus cells. This form of inflammation especially occurs on serous and mucous membranes. It is seen in the healing of wounds, in ulceration, and in the formation of abscesses. (See below.)

Pus—Laudable Pus.—Healthy pus is a thick creamy yellow opaque viscid fluid, with a peculiar sickly smell, an alkaline or neutral reaction, and a specific gravity of 1032. It may be blood-stained, and is very liable to decompose. It consists of:—

(1) LIQUOR PURIS—*Pus Serum*—which is analagous to altered lymph, and contains globulin, peptones, and various products of decomposition—lecithin, cholesterin, fatty acids, and inorganic salts of blood, especially sodium chloride.

(2) PUS CORPUSCLES, which form from 17 to 19 per cent. of pus, are very similar to leucocytes. They are rounded semi-transparent granular masses, and usually have three or more nuclei. In recently discharged pus they show amœboid movements, but these soon cease. They have not, however, like leucocytes, the power of absorbing dead products.

(3) Pus also contains granular masses, changed leucocytes, free nuclei, and albuminous and fatty debris.

Origin of Pus Corpuscles.— One of three modes:—

(1) From Emigrated Leucocytes.—It is improbable that pus corpuscles have their sole source in leucocytes; for, in cases where we have a large formation of pus, it seems difficult to understand how all the pus corpuscles could be produced from leucocytes. The possible explanation is, that white corpuscles themselves are generated more rapidly in cases of suppuration. Again, pus is not usually formed till forty-eight hours after the application of the irritant causing the inflammation; yet, in suppurative inflammation, we find a great increase of leucocytes in the blood; whereas, if they had escaped and formed pus, we should expect fewer in the blood.

(2) From Proliferation of the Connective Tissue Corpuscles.—If an animal be killed and the cornea be then injured and examined, a certain number of bodies like leucocytes are soon found in the lacunæ. Still this does not prove that the corneal corpuscles have divided, for these cells may be derived from leucocytes which have come from the conjunctival sac, or anterior chamber of the eye. In the case of cartilage, they may migrate from the marginal vessels.

(3) From Epithelial Cells.—In the peritoneum, after the application of irritants, we find that the epithelial cells are swollen, enlarged, and multi-nucleated, and show evidence of division. The questions therefore arise—(*a*) Are these multi-nucleated cells changed endothelial cells? or (*b*) are they endothelial cells which have taken up white corpuscles? or (*c*) are they leucocytes which have taken up white corpuscles? They are in all probability endothelial cells which have taken up leucocytes, and which by fission form pus corpuscles. Most probably the pus corpuscles, here as elsewhere, are emigrated leucocytes — though the tissue elements may also aid in their formation.

VIII. Results of Inflammation—

The later processes of inflammation are—

1. Necrosis and Gangrene.
2. Ulceration.
3. Abscess.
4. Repair.

1. Necrosis and Gangrene. (See Section III.)

2. Ulceration.—When suppuration, *i.e.*, the formation of pus, takes place on a free surface there is molecular death of the tissues, and the affected part separates as a slough. An *Ulcer* is a term applied to an open sore produced by loss of substance on the free surface of the skin and mucous membranes in process of ulceration.

3. Abscess.—An abscess is a circumscribed collection of pus—it is a circumscribed suppuration. The leucocytes which have escaped from the vessels gather at the focus of inflammation, and from the pressure caused by them, and from the original injury, as well as from want of nutrition due to thrombosis of the vessels, the tissues undergo softening and liquefaction, and together with the leucocytes die and form pus. For an abscess we require more than mere inflammation, we require the presence of bacteria or of ptomaïnes produced by them; hence, abscesses are rare in internal organs, except from the introduction of septic irritants.

4. Repair. — Later Stages of Inflammation. — When inflammation of a part has ceased, the process of repair begins, a process so closely allied to inflammation that inflammation is by many regarded as reparative in nature, the dilatation of the blood-vessels—hyperæmia—and the leucocytes emigration merely supplying the nutriment necessary for the process.

Highly organised structures such as nerve cells, muscle fibres, gland cells, etc., have little or no power of repair, and such regeneration as does take place is effected by means similar to those by which the ordinary physiological waste is covered. When, therefore, a part has been destroyed, the chief mass of tissue produced as a result of inflammatory repair is formed of connective tissue—the so-called "*Scar tissue.*"

The mode in which this repair by means of connective tissue takes place is two-fold :—

(*a*) The DIRECT METHOD — a process similar to that by which connective tissue was originally developed in the embryo. It may be seen in the cornea after an injury that has not damaged the blood-vessels. The corneal corpuscles swell up, their nuclei divide, the cells send out processes into the damaged part, which is thus soon restored. This mode of repair is rare.

(*b*) The SECOND METHOD OF REPAIR associated with inflammation exhibits two stages :—

(1) Formation of Granulation Tissue.
(2) Extension of Vascular Areas.

In this case the repair is accompanied with an effusion of coagulable lymph — inflammatory exudation — which is replaced by new formed connective tissue. This is at first nourished with blood by new formed blood-vessels—outgrowths of pre-existing vessels.

I. FORMATION OF GRANULATION TISSUE — GRANULATIONS.—Granulations ("proud flesh") is the name given to small masses of cells, like embryonic tissue, which consist of bright red, very vascular nodules or granules, which bleed easily, and which are covered by a fine film of coagulable exudation, with many leucocytes, and cells with two, three, or more nuclei—Pus cells.

Structure.—Granulations in their earliest stage consist of little masses of leucocytes which have escaped from the adjacent vessels, and of small vascular buds, outgrowths from the pre-existing vessels.

In their later stages they are made up of—

(1) Large granular nucleated cells—*fibroblasts*, or fibro-plastic cells.
(2) Many leucocytes.
(3) New formed connective tissue.
(4) New formed blood-vessels.

Now, it is from this granulation tissue that the chief part of repair is effected, the fibroblasts, and possibly the leucocytes, being the active agents.

Fibroblasts—

1. Structure of Fibroblasts.—They are large cells with very granular protoplasm and with a clear, oval, vesicular nucleus, and one or more nucleoli.

2. Origin of Fibroblasts—

(*a*) From *Leucocytes*.—Ziegler maintains that he has seen all stages of transition between leucocytes and fully formed fibroblasts. He constructed a small chamber of two cover glasses, and placed it under the skin of a dog. When examined after some time the space between the glasses was found filled with cells, some of which degenerated; others developed into fibroblasts. The value of this experiment has been called in question, for blood-vessels could grow into the interspace and carry with them connective tissue cells, which might give rise to the epithelioid cells.

(*b*) From *Fixed Tissue Cells*.—There can be little doubt, as seen in the case of the corneal corpuscle above referred to, that connective tissue cells can form new connective tissue corpuscles—fibroblasts, the leucocytes mostly undergoing degeneration.

3. Mode in which the Fibroblasts form New Connective Tissue.—The fibroblasts, which are at first rounded cells, soon shoot out one or more processes and become fusiform or branched cells. These are transformed or give rise to the new connective tissue—

(*a*) Either by a splitting of their protoplasmic processes.

(*b*) Or by a fibrillation of the intercellular matrix, or possibly by both means.

This point is as yet undecided.

II. Extension of Vascular Areas.—The formation of new blood-vessels is an essential factor in the process of repair. The new vessels are formed by outgrowths of pre-existing vessels. They commence as little solid masses of granular protoplasm on the walls of the blood-vessels. These little buds soon become hollowed out, and form vascular channels and loops; the epithelioid cells most likely merely supporting the delicate walls of the new formed vessels. (See "Repair of Wounds," etc.)

The fibroblast may, however, occasionally form new vessels by uniting with pre-existing buds, and then, by a process of vacuolation, they become transformed into vascular channels. These new formed blood-vessels bring blood and leucocytes to the new formed connective tissue. Some of these leucocytes may take part in the process of repair, others are cast off as pus cells.

Section III.

ALTERED CONDITIONS OF NUTRITION.

I. Retrogressive Changes—

1. Degenerations and Infiltrations.
2. Atrophy.
3. Necrosis.
4. Gangrene.

II. Progressive Changes—

1. Repair.
2. Hypertrophy.
3. Tumours.

Section III.

ALTERED CONDITIONS OF NUTRITION.

The life of each organism and of its cell elements depends, amongst other causes, upon the due supply and due assimilation of proper nutriment, and any derangement of this process of nutrition will interfere with the life of the tissues and their elements, and with the proper performance of their functions. These changed conditions of nutrition may be classed as follows:—

Retrogressive—Nutrition impaired or arrested.
Progressive —Nutrition increased.

RETROGRESSIVE CHANGES.

(Nutrition Impaired or Arrested.)

These changes of impaired or arrested nutrition comprise—

(1) Degenerations and Infiltrations.—Necrobiotic changes—*i.e.*, *molecular* death—death of cell elements as distinct from molar death.

(2) Atrophy.—Diminution in size and number of constituent elements of a part.

(3) Necrosis and (4) Gangrene are examples of *molar* death—death of a part of an organism, as distinct from somatic death—death of the entire body.

I. DEGENERATIONS—INFILTRATIONS.

I. Definition—

Degenerations are tissue changes characterised by modifications, chemical and physical, of the constituent elements of the affected parts, resulting in impairment of functions or in death.

Infiltrations, though grouped under a separate heading, are closely allied to degenerations; for, in all cases of infiltration, there is probably an accompanying degeneration.

II. **Division**—The chief forms of Necrobiosis are—

1. Cloudy Swelling.
2. Fatty Degeneration and Infiltration.
3. Caseation.
4. Calcification.
5. Serous Degeneration.
6. Mucoid "
7. Colloid Degeneration.
8. Hyaline "
9. Waxy "
10. Vitreous "
11. Pigmentary "
12. Uratic "
13. Glycogenous "

III. **Nature of the Necrobiotic Process**—

This process may consist—

1. In a simple rearrangement, mechanical or chemical, of the molecules of the tissue elements, as seen in cloudy swelling.

2. In a transformation of the constituents of the cells into some compound usually of a lower type—*e.g.*, fatty degeneration of muscle, etc.

3. Or it may consist in an absorption by the tissue elements of some foreign matter—*e.g.*, uratic degeneration.

4. Or it may be a combination of the cell protoplasm with foreign substances—*e.g.*, waxy degeneration.

IV. **Order**—It will be convenient to discuss each of these degenerations under the following headings:—

1. Definition of the process.
2. Sites or positions in which it occurs.
3. Cause of the change.
4. Characters of the Affected Parts.
   (*a*) *Macroscopic.*
   (*b*) *Microscopic.*
5. Nature of the Change produced.
6. Chemistry of the morbid products.
7. Sequelæ.

I.—CLOUDY SWELLING.

I. DEFINITION — Cloudy Swelling (frequently called albuminous, molecular, granular, or parenchymatous degeneration) is a change in tissue elements, closely associated with high temperatures. It is widespread in its distribution, and often rapid in its development.

II. SITES — Cloudy Swelling usually affects highly organised cells, such as gland cells, heart fibres, muscle, liver, lungs, kidneys, etc.

III. CAUSES—

1. High temperatures as in fevers.
2. Early inflammations.
3. Blood-poisoning.
4. Phosphorus, arsenic, antimony poisoning.

IV. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC. — The tissues become swollen and opaque, more friable, and less vascular than normal.

2. MICROSCOPIC.—Individual cellular elements become irregular in outline, very granular, vacuolated, and swollen. The nuclei of the cells are also enlarged and obscured.

V. NATURE OF THE CHANGE is as yet unknown, but three theories have been brought forward—

1. By some it is said that cloudy swelling is due to the *intra-cellular network* of the affected cells *becoming more evident* than normal.

2. By others that it is due to the precipitation of the *proteids* of the cells by the acids circulating in the blood.

3. That it is a form of hypertrophy of the cells with a tendency to degeneration from excessive nutritive activity.

VI. CHEMICAL CHARACTERS—

1. The granules seen in the cells are probably of an *albuminous* nature, for they disappear on adding acetic acid.

2. They are not *fat* granules, for—

(*a*) They do not stain black with osmic acid.

(*b*) They are not dissolved by chloroform or by ether.

VII. SEQUELÆ—

1. Resolution—in which case the granules disappear, and the cells regain their usual aspect.

2. Interference with the performance of function.

3. Fatty degeneration.

II.—FATTY DEGENERATION.

I. DEFINITION—This degeneration consists in an accumulation of granules of fat in the affected tissues and organs. It is one of the most common forms of degeneration, and occurs alike in living and dead tissues, whether animal or vegetable.

II. DIVISION—The term, fatty degeneration, has been applied to three somewhat different yet allied conditions, viz.:—

1. **Fatty Degeneration proper** or fatty metamorphosis, in which the tissue elements are changed into fat.

2. **Fatty Infiltration.**—Where there is an abnormal amount of fat taken up and stored in the affected cells—the term being used when a few organs only are affected.

3. **Adiposity.**—Where there is an abnormal storage of fat in normal situations, *i.e.*, in connective tissue corpuscles—the term being applied when the body generally is affected.

II. SITES—Fatty degeneration proper is unlimited in its distribution for it may affect almost any part of the body.

III. CAUSES—All cases of imperfect nutrition tend to cause fatty degeneration, hence it is found:—

1. In old age.
2. In dead products.
3. In wasting diseases, such as phthisis.
4. In cases of excessive use—*e.g.*, after hypertrophy.
5. In cases of arrest or defect in supply of oxygen, as seen in phosphorus poisoning, in pernicious anæmia, etc.
6. In certain nerve lesions.
7. After cloudy swelling.

IV. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC.—The affected organ is usually enlarged, its edges are rounded, it is of a creamy yellow colour, more friable than normal, and of a less specific gravity.

2. MICROSCOPIC.—The cells are increased in size, are rounded and full of oil globules. These appear as colourless bright droplets, with a dark outline. These drops may, though they rarely do, run together into larger drops. The nucleus of the cell is also affected; the cell wall may give way and thus perish, and in its place is left a mass of fatty granules and debris.

V. NATURE OF THE CHANGE—Three theories:—

1. That fatty degeneration is due to excessive absorption of fat by the affected cells.

2. That it is due to defective oxidation and elimination of fat.

3. That it is due to a metamorphosis of the protoplasm of the affected cells into fat. This is the most probable hypothesis, for—

(*a*) Carbo-hydrates are one of the chief products of the decomposition of proteids, as well as urea, uric acid, and other by-products.

(*b*) Again, in the ripening of cheese, there is a transformation of proteids into fat.

(*c*) In the formation of adipocere (a change taking place in muscle, etc., when left for some time in contact with water), we have proteids becoming fat.

(*d*) Animals fed on lean meat can store up more fat than can be accounted for by the fat in their food—therefore, they manufacture it from the proteids upon which they feed.

(*e*) In phosphorus poisoning there is a great accumulation of fat in many of the organs of the body. Now, if an animal be fed on non-fatty diet and phosphorus be at the same time administered, the accumulation of fat which takes place cannot come from the food. It must, therefore, have its source in the transformation of the protoplasm of the cells of the affected parts into fat; the phosphorus present preventing the oxidation and removal of the fat thus produced.

VI. CHEMISTRY—Fats are compounds of fatty acids with glycerine. When treated with alkalies they undergo saponification.

Tests—

1. Fat globules are stained black with osmic acid.
2. They are soluble in alcohol and in ether.
3. They are insoluble in acetic acid.
4. They are not stained by carmine, etc.

VII. SEQUELÆ of fatty degeneration are—

1. Resolution—where the fat is removed from the affected cells which regain their normal appearance.
2. Liquefaction and disintegration of the cells.
3. Calcification.
4. Formation of *Cholesterine* and of *Margarin* Crystals.

(*a*) **Margaric Acid,** or fat Crystals, appear as feathery masses, either within the affected cells or amongst their debris.

(*b*) Cholesterine.—This is the only free alcohol in the body. It is a monatomic alcohol, and is found in blood, bile, gall stones, and in the products of fatty degeneration.

Cholesterine Crystals present two forms—

(1) An *anhydrous* form, existing as fine needles.

(2) A *hydrated* form—brilliant, colourless, rhombic plates, notched at the angles.

Tests for Cholesterine—

(1) Strong sulphuric acid and iodine give a violet colour, changing to green, red, and blue.

(2) Cholesterine crystals are insoluble in water and in cold alcohol, but soluble in hot alcohol, ether, and chloroform.

(3) The crystals, dissolved in chloroform and an equal volume of sulphuric acid, give a blood-red solution, changing, when exposed to air, to blue, green, and yellow.

(4) Solution of cholesterine evaporated to dryness with nitric acid, and treated with ammonia, the residue turns red.

FATTY INFILTRATION.

I. Definition—Fatty infiltration differs from fatty degeneration in being not a metamorphosis of the cell protoplasm into fat, but a mere exaggeration of the normal process of the storage of fat. It is, however, usually associated with more or less degeneration.

II. Sites—

1. In liver cells.
2. In the heart, muscle, etc.
3. In connective tissue corpuscles.

III. Causes—

1. Defective oxidation of the fat formed in the body or taken as food—*e.g.*, as a result of sedentary occupations.
2. Excessive supply of fatty and starchy foods.

3. Certain constitutional states—*e.g.*, after child-bearing, after certain fevers, and after castration, there is a tendency to accumulation of fat.

4. Certain nervous lesions give rise to similar excessive storage of fat.

IV. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC.—The organ is enlarged, heavier than normal, though of less specific gravity. It is yellow in colour, and on scraping the cut section, globules of oil are left upon the knife.

2. MICROSCOPIC.—The cells contain fat globules, which are at first small, but afterwards run together into larger drops, and, in so doing, push the nucleus of the affected cells towards the wall.

V. NATURE OF THE CHANGE—Fatty infiltration, as before stated, is an accumulation of fat in the affected cells, especially connective tissue cells; but is commonly accompanied by more or less degeneration.

VI. CHEMISTRY—See "Fatty Degeneration."

VII. SEQUELÆ—

1. Resolution.
2. Impairment of Function.

III.—CASEATION.
(Tyrosis.)

I. DEFINITION—This term is applied to a pathological change in which the affected parts assume a cheesy-looking appearance; the cheesy masses being either (1) hard or (2) soft in consistence.

II. SITES—Caseation affects:—

1. Closely packed groups of cells, such as are seen in the alveoli of the lungs.

2. Infarcts, residues of abscesses, tubercular deposits.

3. Lymphatic glands.

III. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC. — The part becomes more homogeneous, and may be either softer or firmer than normal.

2. MICROSCOPIC. — The caseous *foci* are seen to consist of either a *soft* or *firm* yellow friable, cheesy-looking substance, which is composed of atrophied cells, fatty debris, and cholesterine crystals. A completely caseated mass may be either homogeneous—neither cells nor nuclei remaining —or, if still further advanced, it may consist of a mere mass of granules.

IV. NATURE OF THE CHANGE—

1. Caseation is probably due to a drying up and removal of moisture from already degenerated products.

2. By some it is regarded as a direct change of the affected cells into caseous material.

V. CHEMISTRY—

Caseous matter gives no distinct chemical reactions.

VI. SEQUELÆ—

1. Caseous masses may become calcified, or
2. They may soften, break down, and be absorbed.

IV.—CALCIFICATION.

I. DEFINITION—This degeneration, also called Petrification, consists in a deposition, within the affected tissues and organs, of lime and magnesia salts. The process is closely allied to the deposition of lime salts which takes place in the formation of bone; but in the ossification of bone, the lime salts combine with the protoplasm of the tissues, whereas, in pathological calcification, such a chemical union is rare, the salts, as a rule, merely infiltrating the affected parts.

II. SITES—Calcareous degeneration is chiefly found—

1. In tissues already dead—*e.g.*, in caseous masses, in dead parasites, in dead fœtuses (lithopædia), in tumours, in veins (vein-stones).

2. As a sequel to fatty degeneration.

3. In middle coat of arteries, on mitral and aortic valves, more rarely in veins.

4. In ganglion cells, in the prostate, in cartilage, in tendons.

III. CAUSE AND NATURE OF THE CHANGE—

1. The cause of this degeneration is not as yet determined, but it is probably owing to some chemical affinity in the diseased tissues for lime salts.

2. By some authors it is maintained that the lime and magnesia salts in the body are kept in solution by the carbonic acid in the blood, and this they conclude from the fact that calcareous deposits rarely take place in the walls of veins—venous blood containing more carbonic acid than arterial blood. The reason, however, why calcareous deposits affect arteries rather than veins is due to the fact that the arteries are more liable to injury and to strain than the veins.

IV. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC.—Commencing calcification cannot be recognised by the naked eye, but in advanced cases it gives a white colour and a hard gritty feel to the diseased part.

2. MICROSCOPIC.—Small granules, like grains of dust, are seen in the cells and in the ground substance.

V. CHEMISTRY—

1. The granules consist of calcium and magnesium salts in combination with carbonic and phosphoric acids.

2. If treated with dilute acids, the granules dissolve and the normal tissue reappears.

METASTATIC CALCIFICATION consists in the transference of lime and magnesia salts from one part of the body, and their re-deposition in some other part where they do not normally occur. The process is said to take place in osteo-malacia and in rickets—the secondary deposits in these cases taking place in the kidneys.

V.—SEROUS DEGENERATION.

I. DEFINITION—Serous or dropsical degeneration is closely allied to cloudy swelling, and is, in all probability, not a distinct form of degeneration.

II. SITES—It occurs in the cells of dropsical tissues, and in epithelial cells during inflammation.

III. CHARACTERS OF THE AFFECTED PARTS—Serous degeneration especially affects epithelial cells which imbibe a serous-like fluid, become swollen, translucent, and vacuolated. The granules in the cells are pushed to the periphery, the nucleus swells up and becomes clear.

IV. CAUSE AND NATURE OF THE CHANGE—Serous degeneration arises as a consequence of damage to the nutrition of the cells—*e.g.*, in burns.

V. CHEMISTRY—In stained preparations, dropsical cells remain clearer than the healthy tissue.

VI.—MUCOID DEGENERATION.

I. DEFINITION—The secretion of mucus in mucous glands and on mucous membranes is the physiological type of this degeneration. In certain pathological states, however, we have an excess of this same process of mucus formation, resulting in what is known as mucoid degeneration.

II. SITES—

1. In cells—*e.g.*, in mucous catarrh.

2. In degenerated products of inflammation.

3. In formed tissues — *e.g.*, cartilage, bone, connective tissue, adipose tissue.

4. In new growths—*e.g.*, sarcoma, etc.

These last are rather cases of degradation than of degeneration (see below).

III. CAUSES—Not known.

IV. CHARACTERS OF THE AFFECTED PARTS—

The tissue becomes homogeneous, colourless, translucent, and of a soft jelly-like consistence. The affected cells have a similar appearance.

V. NATURE OF THE CHANGE—

1. It may be a transformation of the protoplasmic elements of the cells or of the intercellular matrix into mucin.

2. It may be an excess of the normal process of the secretion of mucus.

3. In as far as it affects *connective tissue* it is said to be a somewhat different process from that which affects other structures. It is therefore better called *myxomatous degeneration* — the fibrils of the tissue being replaced by a mucoid substance, the cells remaining unaffected. The process is a metaplasia — a degradation from a higher to a lower type of tissue.

VI. CHEMISTRY—

1. Mucus—the chief constituent of which is mucin—is precipitated by acetic acid, alcohol, alum, and by mineral acids.

2. Mucin is closely allied to albumen, but differs from it—

(*a*) In not containing sulphur.
(*b*) In being insoluble in excess of acetic acid.
(*c*) In not being precipitated by boiling, by tannin, or by perchloride of mercury.

Both gelatine and chondrin are precipitated by tannin and by perchloride of mercury.

VII.—COLLOID DEGENERATION.

I. DEFINITION—This degeneration is closely allied to mucoid change, but is limited in its distribution almost entirely to epithelial cells.

II. SITES—

1. In the thyroid body—
   (*a*) As a normal change in old age.
   (*b*) In colloidal goître.
2. In the prostate gland.
3. In the tubules of the kidney.
4. In colloid cancer.

III. CAUSE—Not known.

IV. CHARACTERS OF THE AFFECTED PARTS—

1. MACROSCOPIC.—The part becomes swollen, translucent, and forms a yellow coloured sago-like or glue-like mass.

2. MICROSCOPIC.—The cells contain small homogeneous globules which may unite to form larger masses, and push the nucleus to the side of the cell. Sometimes the cells are destroyed, and thus are formed homogeneous colloid masses in which there are no traces of the original cells.

V. NATURE OF THE CHANGE — Not understood, but probably the colloid material is derived from the epithelial cells.

VI. CHEMISTRY—

1. Colloid matter closely resembles mucin.
2. It is soluble in hot and cold water.
3. It is not precipitated by acetic acid.
4. Nor rendered turbid by alcohol.
5. It is coagulated by tannic acid.

Reactions—

(1) It stains pink with carmine.
(2) It stains blue with aniline colours.

VIII.—HYALINE DEGENERATION.

I. DEFINITION—This degeneration, both in its appearance and in its distribution, bears a close resemblance to amyloid degeneration, to be presently described. It does not, however, give the reaction with iodine, so characteristic of waxy degeneration.

II. SITES—Hyaline degeneration occurs :—

1. In all parts of the walls of the arterioles, except the endothelium.
2. In basement membranes—*e.g.*, Bowman's capsule.
3. In connective tissue corpuscles, and in lymph corpuscles.

III. CAUSES—

1. Intense inflammations, with suppuration.
2. Fevers, blood-poisoning—*e.g.*, diphtheria.

IV. CHARACTERS OF THE AFFECTED PARTS—Tissues affected with hyaline degeneration become homogeneous, translucent, shining, and are highly refractive.

V. NATURE OF THE CHANGE is unknown, but is probably a chemical transformation of the tissues, causing them to have an increased affinity for proteids; this chemical change being produced by the action of bacteria.

VI. CHEMISTRY (see "Waxy Degeneration")—

1. Water, tannic acid, acetic acid, have no effect.
2. Iodine gives a yellow colour.
3. Iodine and sulphuric acid give a green colour.
4. Methy-aniline violet gives a blue colour.
5. Picrocarmine gives a yellow or pinkish reaction.
6. Logwood gives a dark-blue colour.
7. Millon's reagent (nitrate and nitrite of mercury) gives a reaction very similar to that with proteids.

IX.—WAXY DEGENERATION.

(Amyloid, Albuminous, and Lardaceous.)

I. DEFINITION — Waxy degeneration is a progressive change which tends to affect connective tissues. Widespread in its distribution, and of very common occurrence, it is often sudden in its invasion, coming on in the course of a very short time.

II. SITES—Waxy degeneration may occur in almost any tissue or organ, but is most usually met with in the spleen, liver, kidney, intestine, and lymphatic glands, It is rare in the muscles, brain, etc.

III. CAUSES—

1. Long-standing suppuration — *e.g.*, in chronic abscesses due to bone disease.
2. Chronic phthisis.
3. Constitutional syphilis, even when there is no suppuration, and no bone disease.

IV. Characters of the Affected Part—

1. Macroscopic.—To the naked eye the affected parts appear homogeneous, semi-translucent, and pale in colour, with a smooth, waxy, lardaceous, bacony look. The organs affected are elastic and increased in size, weight, and specific gravity, and offer great resistance to decomposition.

2. Microscopic.—Affected parts have a uniform, translucent appearance, and iodine staining shows that the seat of the change is—

(*a*) The middle coat of arterioles.
(*b*) The entire walls of capillaries.
(*c*) Basement membranes.
(*d*) Filaments of connective tissue—*e.g.*, trabeculæ of lymphatic glands and spleen; perimysium internum and externum of muscle.

Whether connective tissue and endothelium cells are also affected is doubtful.

V. Nature of the Change—

1. It may be a further stage of hyaline degeneration.

2. It may be due to the action of a ferment causing an increased capacity of the affected tissue for the absorption of proteids.

3. It may be due to the union of an albuminous substance in the blood with some constituent of the affected tissue.

VI. Chemistry—

1. Waxy material was at first regarded as a non-nitrogenous substance of a starchy nature, hence the name "Amyloid Degeneration."

2. Most probably it is nitrogenous in nature, closely allied to proteids.

3. Lardacein.—By means of artificial digestion waxy matter yields a substance called Lardacein, which has the composition $C_{53\cdot6}H_{7\cdot0}N_{15\cdot5}O_{22\cdot5}S_{1\cdot3}$

Reactions of Lardacein—

(1) Lardacein is soluble in alkalies, and in strong hydrochloric acid, and this solution diluted with water gives a precipitate of syntonin.

(2) Boiled with sulphuric acid, lardacein gives a violet-coloured solution.

(3) With strong sulphuric acid, it decomposes into leucin and tyrosin.

(4) With eosin, the waxy matter stains bright red.

(5) With iodine, it gives a mahogany brown colour.

(6) Iodine and sulphuric acid give a green-blue or violet colour.

(7) Methyl-aniline violet gives a ruby-red colour, with waxy matter; a blue colour, with healthy tissues.

(8) Iodine green gives pink with the waxy matter, and blue-green with normal structures.

(9) Osmic acid gives no reaction with waxy matter.

TABLE OF REACTIONS.

	Hyaline.	Waxy.
1. Iodine gives . . .	Yellow.	Brown.
2. Iodine and Sulphuric Acid	Green.	Blue.
3. Methyl-aniline Violet .	Blue.	Rose Red.
4. Picrocarmine . . .	Yellow or Pink.	Yellow.
5. Logwood	Dark Blue.	Pale Blue.

VII. SEQUELÆ—

1. Resolution.
2. Fatty degeneration.
3. Calcification (rare).
4. Necrosis, resulting in a mass of granules which do not give the waxy reactions.

Localised Waxy Degeneration.—Over and above the diffuse form of waxy degeneration above described, there exists a localised form, which occurs either as circumscribed masses or as amyloid concretions—the so-called

corpora amylacea. It is met with only in those tissues which are already undergoing morbid change; and hence it especially occurs after inflammation, and round old abscesses. By some it is considered to have its origin in the absorption of poisons generated during the process of suppuration. Localised waxy degeneration is also seen in lymphatic glands, in degenerating cartilage, and in various tumours.

Corpora Amylacea are supposed to be masses of waxy matter. They are found normally in the central nervous system; in the choroid plexus and meninges of the brain; in the prostate; in old blood clots; in cancers; and in ducts of glands, etc.

STRUCTURE.—Corpora amylacea have a peculiar laminated appearance, their centre often containing blood pigment. Their mode of origin is not known, but it is stated that they are due to the metamorphosis of the albumen of the blood and of epithelial cells. This, however, is denied by some. In the prostate, corpora amylacea often tend to calcify.

CHEMISTRY.—The exact chemical composition of amyloid concretions is unknown.

(1) Iodine stains them dark brown.
(2) Iodine and sulphuric acid gives a brown or blue colour.
(3) Methyl-aniline violet gives the same reaction as waxy matter.

X.—VITREOUS DEGENERATION.

(Zenker's Degeneration.)

I. DEFINITION—Vitreous degeneration occurs in the muscles which have been exposed to severe strain, injury, or inflammation; hence it occurs after fevers. Thus, after typhoid fever, it is seen in the muscles of the abdominal wall.

II. CHARACTERS OF THE AFFECTED PARTS—There is no well marked naked-eye change, though the muscles are somewhat semi-opaque, paler, and more homogeneous than normal. Under the microscope, the muscular fibres have a swollen, vitreous, or glassy appearance, and the fibres show a tendency to split transversely.

III. NATURE OF THE CHANGE—

1. Vitreous degeneration is by some regarded as similar to colloid degeneration.

2. By others it is regarded as due to the coagulation of the myosin—a coagulative necrosis.

3. It may be analogous to Hyaline or to Waxy Degeneration.

IV. CHEMISTRY—The affected parts stain pink with picrocarmine.

XI.—PIGMENTARY DEGENERATION.

I. DEFINITION—Pigmentary degeneration and infiltration consists in the deposit in the affected tissue of pigment in abnormal amount.

II. DIVISION—Pigmentary degenerations may be classed under the following headings:—

1. Pigments derived from without—extraneous or false pigmentation.

2. Pigmentation due to the local action of various chemical substances.

3. Pigmentation due to pigments formed in the body, and may occur—

(1) By direct chemical transformation of—

(*a*) Blood pigment—Hæmatogeneous Pigmentation,

(*b*) Bile pigment—Hepatogeneous Pigmentation; or

(2) By the nutritive activity of the cells of the affected tissue, and is subdivided into—

(*a*) True pigmentary degeneration.

(*b*) Derangement in the distribution and quantity of normal pigments.

(*c*) Pigmentary new growths.

1. Pigments derived from without—

Extraneous Pigmentation is due to the entrance from without of various substances which are deposited in the cells and tissues—*e.g.*,

(1) Argyria—*i.e.*, pigmentation due to long use of compounds of silver. It causes a brown colouring of the skin and internal organs.

(2) Coal-Miners' Lung—pigmentation due to coal-dust.

(3) Tattooing, etc.

Mode of Entrance of the Pigments—

(1) Through the *air passages*, thence to the *lymphatics* directly, or through the *air cells* of the alveoli, which, owing to the irritation set up by the presence of a foreign substance, proliferate, become multi-nucleated, take up the pigment particles, and pass them on into the lymphatics and lymphatic glands.

(2) Through *wounds in the skin*—*e.g.*, tattooing.

(3) Through the *stomach*, as in Argyria and Lead-staining.

2. Pigmentation due to the action of Chemical Agents—

(1) H_2SO_4 gives a black stain.

(2) HNO_3, a yellow or bright green coloration.

(3) Post-mortem stains are of a green, bluish-black colour, and are due to the combination of sulphuretted hydrogen and iron compounds.

(4) The changes which take place in blood, post-mortem, are similar to those produced by artificial means.

3. Pigmentation due to Pigments formed in the Body—

I. (*a*) Hæmatogeneous Pigmentation—Pigmentation due to the Direct Chemical Transformation of the Blood Pigment.—Most of the pigments found in the body have this source. They are formed by the splitting up of the hæmoglobin of the blood.

This may take place—

1. *While the blood is still circulating*, and is called Malanæmia—a condition seen in malaria fever, etc., in which the liver, spleen, and brain become pigmented. The change is probably due to the action of micro-organisms.

2. *In escaped blood—i.e.*, in blood clots—the change in this case consisting—

(*a*) In one part of the red corpuscles and plasma being taken up and removed unchanged by the lymphatics.

(*b*) In some of the corpuscles breaking up, and the hæmoglobin becoming diffused in the surrounding tissues—well seen in subcutaneous extravasations of blood.

(*c*) Part of the absorbed pigment forms crystals in the tissue—hæmatoidin crystals, which consist of either ruby red rhombic plates or crystalline needles.

(*d*) Part of the disintegrated corpuscles and pigment are taken up by leucocytes, and carried to other parts of the system, while part remains *in situ*, forming granular or crystalline masses, causing local discoloration.

Nature of the Pigment—

1. The pigment may be yellow, brown, or black granules.
2. It may form crystalloids; or

3. Actual crystals, which are of two forms—

(*a*) Ruby-red rhombic plates.

(*b*) Yellow or brown needles.

Chemical Characters and Relations of Blood Pigment—

1. The chief blood pigment is a proteid, Hæmoglobin, which consists—

(*a*) Of a coloured compound, *Hæmatin*, which contains all the iron of the hæmoglobin; and

(*b*) Of a colourless proteid, *Globin* or *Globulin*.

2. Hæmoglobin, when treated with chemical reagents, such as potash, etc., at once splits up into these two constituents.

3. Hæmoglobin, with glacial acetic acid and common salt, forms minute rhombic crystals of a substance called *Hæmin*, said to be the hydrochlorate of Hæmatin.

Changes in these Blood Pigments in Pathological Conditions.—The hæmoglobin changes to a substance called *Hæmatoidin*. The first step in this transformation is the breaking up of the hæmoglobin into globin (or globulin) and the coloured compound, *Hæmatin*, which, as before stated, contains all the iron of the original hæmoglobin. This iron may be set free from the hæmatin and be deposited in the tissues as the hydrated ferrous oxide, the presence of which can be detected—

(*a*) By hydrochloric acid and ferrocyanide of potassium, giving a dark blue colour (Prussian blue), and

(*b*) By ammonia hydrogen sulphide (NH_4HS), giving a black colour.

The coloured non-proteid residue of hæmatin is changed to Hæmatoidin—a substance which contains no iron, and which has not been produced artificially. It is said to be formed by the action of certain ferments, such as pancreatin, etc.

Characters and Reactions of Hæmatoidin—

1. Hæmatoidin occurs—

(1) As granules—*size* of which varies—*shape*, round or irregular—*colour*, yellow, red, brown, or black.

(2) As crystals—

(*a*) Opaque rhombic plates, with a yellow or ruby-red colour.

(*b*) Fine needles or plates, more or less transparent and glistening.

2. Hæmatoidin is insoluble in alcohol, ether, dilute acids, and alkalies.

3. It is soluble in carbon di-sulphide and in chloroform.

4. With strong caustic potash the crystals break up into granules and dissolve.

5. Strong sulphuric acid gives a yellow colour, and a play of colours.

6. Nitrous and nitric acid give a play of colours.

7. Not only in the form of its crystals, but also in its chemical reactions hæmatodin closely resembles bilirubin, the chief bile pigment.

I. (*b*) Hepatogenous Pigmentation — Pigmentation due to the direct transformation of Bile Pigment.—The pigments of the bile are closely related if not identical with those of the blood.

If red corpuscles be injected into the circulation, they break up, and an excess of pigment is found in the liver and in the urine.

After long standing jaundice, bile pigment is deposited in the kidneys and subcutaneous connective tissues. The pigment occurs as orange-yellow crystalline granules.

Xanthelasma — the creamy yellow patches seen below the eyes and in the flexure aspect of joint in jaundice — is probably due to fatty degeneration of the cells in these regions, not to true bile pigmentation.

II. Nutritive Form of Pigmentation.

(*a*) *True Pigmentary Degeneration.*— In heart muscle there is a certain amount of pigmentation existing normally, but under certain conditions, due to the decreased nutritive activity of the muscle cells, we get an excessive deposit of pigment, causing brown atrophy of the heart. Similar changes are also seen in liver cells and in ganglion cells.

(*b*) *Derangement in the Quantity and Distribution of Normal Pigments.*— This occurs in Addison's disease, in which we find a marked bronze colouring of the skin, especially in those regions where pigment exists normally— *e.g.*, in the axilla, in mucous membranes, in the skin, etc.

(*c*) *Pigmented New Growth.*—The pigment in the case of new growths differs from the pigment derived from the blood, in having a higher per cent. of carbon, and in containing sulphur. It occurs as fine dark brown coloured granules, which are—

(1) Insoluble in alcohol, in ether, in dilute acids, or alkalies.

(2) Soluble in boiling alcohol, and in strong acids and alkalies.

(3) Solutions in potash are discoloured by chlorine.

XII.—URATIC DEGENERATION.

I. Definition — Uratic Degeneration consists in a deposit of Urates, and of Carbonates and Phosphates of Soda, in the affected tissues.

II. Sites—

Joints, ligaments, cartilage, fibrous tissue, fascia, tendons, heart valves, endocardium, arteries, skin, kidneys.

III. CAUSES—

1. May be due to breaking down of the tissues.
2. Or to defective oxidation.
3. Or to defective metabolism and elimination.

IV. CHARACTERS OF THE AFFECTED PARTS—

In the affected tissues are found needle-shaped crystals or shining amorphous masses called tophi—chalk-stones.

V. NATURE OF THE CHANGE—

Most likely a degeneration, not a mere infiltration, for there is invariably a diseased state of the tissues before the uratic deposit occurs.

The salts are first deposited in the cells and afterwards in the matrix of the tissues, whereas calcification begins in the matrix.

VI. CHEMISTRY—

1. Uratic degeneration is distinguished from calcification by the fact that the calcareous deposit is dissolved by dilute acids such as hydrochloric acid, etc.; whereas the deposits of urates at first decompose and disappear on the addition of the acid, but are afterwards re-deposited as uric acid.

2. With nitric and nitrous acid and ammonia, the urates give a purple colour.

XIII.—GLYCOGENOUS DEGENERATION.

Glycogenous degeneration is a condition found in the liver in some cases of diabetes. In this disease there is an excessive transformation of glycogen and increased excretion of glucose, and a deposit of a substance of the nature of glycogen in the liver cells and in the tubules (especially the looped tubules of Henle) of the kidney.

The epithelial cells become swollen, homogeneous, and hyaline, but the outlines of the cells are usually retained.

II.—ATROPHY.

I. DEFINITION—

Atrophy consists in the diminution in the size or in the number of the essential elements of a part, without any marked change in their chemical or physical characters.

The term Atrophy, therefore, does not apply where there is a chemical or physical change.

Atrophy must not be confounded with Hypoplasia or with Aplasia, terms which express absence, or incomplete development, of a part.

Moreover, there may be an actual increase in size of the affected parts, though at the same time there is an actual decrease in the essential constituents—*e.g.*, in emphysema of the lung, the actual size of the lung is increased, whereas the lung tissue has decreased.

II. DIVISION—

1. (*a*) GENERAL ATROPHY—That which affects the whole body, as seen in old age.

   (*b*) LOCAL ATROPHY—That which affects particular tissues and organs—*e.g.*, liver, etc.

2. PHYSIOLOGICAL ATROPHY—in which there is a wasting of the tissue affected, due to—

   (*a*) *Loss of Function*—*e.g.*, in thymus gland.

   (*b*) *Retrogression*—*e.g.*, atrophy of bone in old age.

   (*c*) *Involution* of parts—*e.g.*, mamma and uterus after climateric period.

3. PATHOLOGICAL ATROPHY—

   (*a*) *Simple Atrophy*—where there is a diminution in the size of a tissue or organ, due to loss of *size* of its constituent elements.

(*b*) *Numerical Atrophy*—diminution in size, due to loss in *number* of the constituent elements.

These are rarely cases of mere atrophy, there being usually an accompanying degenerative process.

(*c*) *Degenerative Atrophy*—where there is a marked degeneration concomitant with the atrophy—*e.g.*, in brown atrophy of the heart.

(*d*) *Sclerotic Atrophy*—in which there is an increase in the fibrous tissue, with loss of the functional elements of the affected part.

(*e*) *Absorptive Atrophy*—in which the process is attended with removal by absorption of the affected part.

III. Causes of Atrophy—

1. Defective Nutrition—

(*a*) Defect in the quantity and quality of the supply of nutriment may be—(1) General, as seen in inanition; or (2) Local, as seen in the obstruction of an artery.

(*b*) Defect in the assimilating powers of the affected part, due to—(1) Exhausted vitality. (2) Overwork—*e.g.*, hypertrophy is often followed by atrophy.

2. Defective Functional Activity. — Under this category comes Physiological Atrophy; hence, the change is seen—

(*a*) In muscles which are not used.

(*b*) In bones, when part of a limb has been removed.

(*c*) In nerves—*e.g.*, optic, after removal of the eyeball.

(*d*) In glands, when their ducts are obstructed.

3. Pressure.—This is another great cause of atrophy. Constant pressure leads to atrophy; an intermittent pressure, to hypertrophy. This pressure may be—

(*a*) *External* — from without — *e.g.*, pressure of a tumour, etc.

(*b*) *Internal* — from within — *e.g.*, hydronephrosis, emphysema.

4. Neurotrophic.—Atrophy due to nerve lesions.

III.—NECROSIS.

(Molar Death.)

I. Definition—

Necrosis—molar or local death—may be defined as the death of a part of an organ or tissue as distinguished from the death of the whole organism (somatic death), or from death of the individual elements (molecular death).

II. Causes—

1. Direct Causes—

(*a*) *Mechanical*—such as arrest the vascular supply of parts—*e.g.*, ligatures, etc.

(*b*) *Chemical* — *e.g.*, various poisons which kill the tissues.

(*c*) *Thermal*—intense heat or cold—*e.g.*, frost bites, etc.

(*d*) Intense inflammations—*e.g.*, dysentery, typhoid, etc.

2. Indirect or Predisposing Causes—

(*a*) Weakness of the general vitality—*e.g.*, in progressive muscular atrophy, etc.

(*b*) Weakness of the circulation.

(*c*) Certain nerve lesions — *e.g.*, diseases of spinal cord cause bed sores.

III. Division—

The various kinds of necrosis are really further stages of the process of necrosis. They are :—

1. Coagulative Necrosis.—Necrosis accompanied by coagulation, the dead cells becoming solid and firm, and uniting into a homogeneous mass.

2. Colliquative Necrosis.—Necrosis in which the affected tissue elements liquify; the cells absorb the liquid by which they are surrounded; their protoplasm dissolves; the cells break up, leaving a residue of proteid granules, fat globules, fat crystals, cholesterine, crystals from changed blood pigment, and granules of carbonate of lime.

IV. Results of Necrosis—

1. Regeneration of the tissue, in which case the affected part is replaced by new growth.
2. The dead part may become gangrenous.
3. May caseate or calcify.
4. Formation of cysts, due to absorption of the dead products.

IV.—GANGRENE.

I. Definition—Gangrene may be defined as necrosis accompanied by decomposition.

II. Division—

1. Primary Gangrene — viz., that form of gangrene which starts, *de novo*, without previous necrosis.

2. Secondary Gangrene—that form of gangrene consequent on previous necrosis.

According to the changes which take place in the affected part, each form is subdivided into—

(1) Moist Gangrene.
(2) Dry Gangrene.

III. CAUSES—

1. PREDISPOSING CAUSES. — Anything that lowers the vitality of a part—*e.g.*,

(*a*) Old age.

(*b*) Feeble heart action.

(*c*) Deterioration in blood, as in Bright's disease.

(*d*) Injury to the nervous system.

2. EXCITING CAUSES:—

(*a*) Inflammation, especially septic.

(*b*) Physical and Chemical agents which cause damage to a part.

(*c*) Obstruction to arterial and venous circulation.

IV. CHARACTERS OF THE AFFECTED PART—

1. Moist Gangrene — SPHACELUS.— In this form of gangrene the blood is rapidly diffused through the walls of the blood-vessels, the part becomes moist and soft, the colour changes rapidly from green to black, the surface epithelium is shed, and blebs with gas and fluid are formed, giving rise to a peculiar crepitant feeling (gangrenous emphysema). There is rapid destruction of the soft parts, with the formation of a grey black semi-fluid mass with the peculiar smell of decomposing animal matter, and containing the remnants of the original tissue elements and various products of decomposition, such as margarin crystals, triple phosphates, and granules of brown or black pigment. Moist gangrene tends to spread rapidly, and, owing to the absorption of the dead products, tends to produce blood-poisoning.

Between the living tissue and the gangrenous part is formed a bright red line—line of demarcation—this deepens and the dead part ultimately separates as a slough or sphacelus from the living tissue, and a healthy granulating wound is left, which ultimately cicatrises.

Examples of Moist Gangrene—

(*a*) Inflammatory gangrene.
(*b*) Traumatic gangrene.
(*c*) Hospital gangrene.
(*d*) Diabetic gangrene.
(*e*) Cancrum oris.
(*f*) Carbuncle.
(*g*) Bedsores, etc.

2. Dry Gangrene—Mummification.—In this form of gangrene—of which senile gangrene may be taken as a typical example—the affected part becomes at first engorged with blood, then pale, cold, tallow-like. It now rapidly changes colour, due to changes in the effused blood, and it has an offensive odour. The fluid then evaporates, the part begins to shrivel, gets brown or black, dry, hard, and brittle; the cellular elements disintegrating, but the bones, tendons, and cartilage remaining. At the same time products of decomposition are formed, viz., compounds of the fatty acids, ammonia sulphide, sulphuretted hydrogen, etc.

A zone of inflammation is left between the living and dead tissues, as in the moist form, and the part ultimately separates.

Examples of Dry Gangrene—

(*a*) Senile gangrene.
(*b*) Gangrene from frost bites.
(*c*) Gangrene from emboli or ligature of arteries.
(*d*) Raynaud's disease.

PROGRESSIVE CHANGES.

(Nutrition Increased.)

These progressive changes of nutrition are characterised by increased cell formation, and increased activity of growth. They result—

1. Either in the repair or regeneration of lost parts.
2. Or in the overgrowth of parts or organs beyond their normal limits.
3. Or in the production of new growths.

Hence these changes may be considered under the following headings:—

(1) Repair.
(2) Hypertrophy.
(3) Tumours, or Neoplasia.

I. REPAIR.

I. Definition—

Repair consists in the regeneration, or replacement by new formed tissue, of parts that have been destroyed by the various retrogressive processes.

II. General Laws of Repair—

These laws govern not only the process of repair, but also the growth of tumours and the process of Hypertrophy. They are as follows:—

1. The regeneration or reproduction of the new tissue takes place from tissues of the same embryonic layer as that from which the already existing tissue originated.

2. The new formed tissue belongs to the same type as that from which it originates.
3. The occurrence of repair is conditional upon proper supply of nutriment.

New formed tissues may, however, sometimes be modified according to circumstances; thus, they may remain more embryonic in type than the corresponding normal tissue. Occasionally they may be more complex; thus, an epithelium more complex than the original may be produced, but the usual tendency is to the formation of more simple types.

III. Order—

We shall consider—

1. Repair in Vessels—
   - (1) After rupture.
   - (2) After ligature.
2. Repair in Wounds—
   - (1) By direct union.
   - (2) By granulation tissue.
3. Repair of Individual Tissues.

I. Repair in Vessels—

1. After Rupture.—When an artery has been severed or ruptured, hæmorrhage proportionate to the size and number of the injured vessels is the immediate result; after a time the hæmorrhage ceases, or is arrested by a process very similar to that which takes place in the torsion of a vessel. The ruptured inner and middle coats retract and curl up within the lumen of the vessel, and thus partially occlude it. A clot now forms at the injured spot, the damaged inner coat acting like a foreign body to the

blood, causes a deposition of blood plates and consequent coagulation (see page 18). Then, by the process described in the following section, the occluded vessel becomes converted into a fibrous cord, and is thus permanently closed.

2. After Ligature.—If an artery be ligatured in its continuity, the inner and middle coats are cut through by the ligature, and curl up and retract within the lumen of the vessel; the external coat is merely crumpled up by the ligature. Next, a thrombus forms on both the distal and proximal side of the ligature; the proximal thrombus being the firmer and the better marked. It is conical in shape—its base at the point of ligature, its apex free—and reaches to the next collateral branch. The distal thrombus is, on the other hand, smaller, and not so firm, because the collateral circulation reaches the distal part of the vessel and disturbs the formation of the clot. The thrombus thus formed becomes more or less adherent to the vascular wall, and, by a process known as the "*Organisation of Thrombus*," is gradually replaced by vascularised fibrous tissue. By this means the occluded part of the vessel is converted into a fibrous cord, and becomes adherent to the surrounding tissues. The remains of the thrombus either caseates or calcifies.

Organisation of Thrombus—

By the organisation of thrombus is understood its replacement by or transformation into a mass of vascularised fibrous tissue. There are two views as to the manner in which this occurs:—

1. According to one view, the process is effected by means of the leucocytes in the thrombus and those derived from the neighbouring vessels. These leucocytes become transformed into fibroplastic cells—fibroblasts—which in turn develop into or give rise to fibrous tissue. The thrombus itself remains passive. The vascularisation of the clot

is carried on by means of new vessels, which are outgrowths of the vasa vasorum. (See "Formation of Blood-vessels," page 93.)

2. According to the second view, the leucocytes play little or no part in the process, the *endothelial cells* lining the inner coat being regarded as the chief agents in the organisation. The vascularisation is accomplished, in this case also, by outgrowths from the vasa vasorum. The thrombus, too, remains passive, and is absorbed by the agency of leucocytes or other multi-nucleated cells found in the situation.

This second view is the more probable, and the one here described in detail.

Details of the Process—

When a vessel that has been ligatured is examined a few hours after the application of the ligature, it will be seen that the thrombus formed at the point of ligature has become somewhat adherent to the vascular walls. As a result of this we find inflammation and irritative reaction of the walls of the vessel, especially of the inner coat. This reaction of the vascular walls may be arranged in three stages:—

(1) Proliferation of the endothelial lining of the inner coat.

(2) Proliferation of all the elements of the inner coat.

(3) Reaction of the entire vascular wall.

(1) The first step, therefore, in the organisation consists in the proliferation of the endothelial cells lining the inner coat. They send out processes into the clot, or form masses which push it aside. At the margins of the clot the endothelial cells grow over the surface of the thrombus.

(2) Secondly, the fibrous elements of the tunica intima are replaced by a gelatinous mass connected with the inner coat; for the connective tissue cells swell up, the fibres soften, and the whole inner coat becomes a mass of granu-

lation tissue, which projects as buds into the thrombus or pushes the clot aside. It also spreads some distance along the vascular wall beyond the clot. The new formed vessels of the granulation tissue are outgrowths of the vasa vasorum.

(3) Finally, the reaction of the entire vascular wall commences with the engorgement of the vessels of the outer coat—branches of the vasa vasorum penetrating the inner coat. Buds consisting not only of granulation tissue, but containing open spaces—*i.e.*, new formed vessels—are seen growing towards the thrombus. Masses of blood crystals, pigment granules, and large, oval, very granular cells, some of which are multi-nucleated, occupy the site of the thrombus, while large flat cells are observed between the apex of the buds and the clot.

The middle coat probably takes no active part in the process.

Thus we find almost the entire coat of the vessel replaced by granulation tissue. These masses of granulation tissue are transformed into fibrous tissue, the clot being removed by means of leucocytes, or by the multi-nucleated cells above referred to. This new formed fibrous tissue contracts and strangulates the new formed vessels. Thus the original vessel becomes permanently closed, and what was formerly an artery becomes an impervious cord.

In some cases there is said to be a canalisation of the thrombus from the lumen of the vessel, buds from the channel of the vessel growing into the thrombus. These buds are then transformed into new vessels which communicate with those from the vasa vasorum.

Rate of the Process—

The reaction of the inner coat takes place early—the proliferation of the endothelial cells occurring in two or three days. The buds are said to be found on the sixth or seventh day, and complete closure of the vessel is accomplished in from two to three weeks.

II. Repair of Wounds—

Healing of wounds takes place in one of two chief ways, know respectively as—

(1) Healing by First Intention.

(2) Healing by Second Intention, or by Granulation Tissue.

From a pathological point of view these two modes of healing are essentially the same, the two processes differing merely in their rate of occurrence, and in the amount of exudation, and of granulation tissue formed.

1. Healing by First Intention—

By this we understand an apparent direct union of the cut surfaces of a wound without the evident formation of granulation tissue. The process occurs in simple incised wounds through the skin and subcutaneous structures, where there is little irritation.

The essentials of the process are a simple or adhesive inflammation of the opposed surfaces of the wound without suppuration. At first, after the infliction of the injury, the wound gapes and there is more or less hæmorrhage from the divided vessels. This soon ceases, and if the edges of the wound be kept in contact and free from irritation, the healing process commences. A quantity of coagulable lymph is poured out between the opposed surfaces of the wound, uniting them together. Vessels of new formation, outgrowths from pre-existing vessels, form vascular loops, and shooting into the coagulated lymph unite with their fellows of the opposite side. New formed connective tissue is now developed from the leucocytes, or form the permanent elements of the tissue, or from both, and gives rise to young vascular connective tissue—Granulation Tissue. This granulation tissue, as we have seen in treating of inflammation, consisted of little vascular buds, leucocytes, and epithelioid cells, called fibroplastic cells. This granulation

tissue ultimately develops into permanent fibrous tissue, which fills up the gap, leaving a white scar to mark the site of the original wound.

Details of the Process—

If a series of simple incised wounds be examined at various periods of time after they have been inflicted, we may observe the following stages in the process of repair:—

(1) *Twelve Hours after the Injury.*—When the initial hæmorrhage, or oozing from the vessels and capillaries, has ceased, we find that the edges of the wound are perhaps slightly vascular and more or less swollen; the swelling, according to one view, being due to engorgement of the vessels—according to another and more probable view, to the loss of elasticity in the tissues. The swelling and redness, however, soon subside, and the gap between the cut surfaces becomes filled up by an exudation of lymph and leucocytes, and a few red corpuscles. These latter, however, soon disappear, and leave the exudation, which was at first of a reddish hue, yellow or almost colourless.

In the immediate neighbourhood of the wound, stasis and coagulation occur in the smaller vessels and capillaries, bringing about the cessation of the hæmorrhage above referred to. This may be called the area of thrombosis.

Outside this area of thrombosis is a zone of retarded blood-flow; exudation of serum and escape of leucocytes. These pass through the cut lymph spaces to the raw surfaces of the wound, and form a layer of coagulable lymph between its opposed edges. As yet there is no congestion of the vessels and no marked change in the surrounding tissues.

(2) *In Twenty-four Hours.* — The fibrinous exudation still continues, and at the surface of the wound presents a laminated appearance. The edges of the wound are infiltrated with leucocytes which still continue to penetrate the coagulable lymph. There is great congestion of the vessels

at the margin of the wound, and this congestion extends some distance beyond the incision, but as yet there is no reaction of the surrounding tissue elements.

(3) *In Thirty-six Hours.*—About this time commences the formation of new capillary blood-vessels. These new formed capillaries begin as outgrowths from pre-existing vessels, and form loops which force their way to the surface of the wound and penetrate the coagulated lymph. Growing across the interval between the cut surfaces, they unite with similar vascular loops from the opposite surface, thus re-establishing the circulation across the gap, and causing the disappearance of the faint blush of redness at first seen at the edges of the wound. The lymph is now dissolved and subsequently removed. Thus is accomplished the vascularisation of the uniting layer of cells. The tissue round the vessels now commences to proliferate, and the connective tissue corpuscles, and possibly the leucocytes, give rise to masses of young vascular connective tissue called granulation tissue.

(4) *In Forty-eight Hours.*—The inflammatory engorgement of the surrounding vessels begins to subside; the exudation of lymph and leucocytes, though less abundant, still continues, and the larger vessels by organisation of their thrombi, become completely closed. At the surface of the wound the epithelial cells begin to grow over the injured area, while in the deeper parts of the wound there is active transformation of the granulation tissue into permanent fibrous tissue.

(5) *In Five Days.*—At this stage masses of embryonic connective tissue, consisting of the interlacing fibres of branched connective tissue cells, fill up the deeper parts of the wound, while at the surface vascular loops, with connective tissue corpuscles arranged at right angles to them, shoot across the interval between the opposed surfaces of the wound. Finally, the fully formed connective tissue begins to contract, causing obliteration of some of the new formed

vessels, and the scar tissue thus formed alone remains as a thin white streak to mark the situation of the original wound.

2. Healing by Second Intention—by Granulation Tissue—

When the edges of the wound are not kept in accurate apposition, or when from any cause healing by first intention is prevented, then we get repair by second intention—*i.e.*, by means of granulations. This mode of repair is the one which always occurs when there has been any great loss of substance.

Details of the Process—

(1) *In Twenty-four Hours.*—As in healing by first intention, so in this case also, there is at first free hæmorrhage, which after a time ceases through thrombosis of the vessels. The inflammatory redness and swelling of the edges of the wound are very distinct, and do not subside, but increase. Many of the leucocytes break down and form pus corpuscles and are discharged from the surface of the wound. Coagulable lymph is poured out on the raw surfaces, and new formed vessels grow out from the pre-existing vessels as capillary loops which shoot out amongst the small round cells which form the superficial covering of the cut surfaces.

(2) *In Forty-eight Hours.*—The tissues round the wound become more sodden, the muscles are paler, and the exudation becomes of a yellowish colour.

(3) *In Three Days.*—The secretion on the raw surfaces is now more purulent, and the vascular loops, with leucocytes and embryonic connective tissue, form small vascular elevations — granulations — which gradually coalesce, and soon cover the whole surface of the wound. These granulations are then transformed into new formed fibrous tissue which, as in the previous case, contracts, obliterating some of the vessels and forming scar tissue.

At the surface of the wound, the epithelium grows over the injured area. Hair and glands, however, are not renewed.

Summary.—Thus we see that in the healing of wounds the chief phenomena are—

1. Hæmorrhage.

2. Exudation of coagulable lymph. This acts as a temporary agent, uniting together the cut surfaces, but does not organise into new fibrous tissue but is gradually removed.

3. Formation of new vessels from pre-existing vessels, leading to—

4. The production of masses of vascular embryonic connective tissue—granulation tissue—which consists of:—

(1) New blood-vessels.

(2) Fibrous tissue corpuscles.

(3) Leucocytes.

(4) Fibroblasts.

5. This granulation tissue ultimately develops into the fully formed fibrous tissue known as *Scar tissue.*

III. Repair of Special Tissues—

The special tissues are repaired in the same manner in which the original normal tissues are produced. Their individual power of repair, however, varies much: some, such as ganglion cells, not being regenerated when once destroyed; while others, such as connective tissue, have the capacity of regeneration in a marked degree.

1. White Fibrous Tissue can easily be reproduced. The new tissue is of a more simple character than the normal tissue, consisting principally of simple fibrillæ arranged in bundles.

2. Yellow Elastic Tissue can also be reproduced, as seen in case of certain myxomatous tumours. Scar tissue, however, is very inelastic.

3. Mucoid Connective Tissue is not very readily reproduced.

4. Adipose Tissue can be repaired to a large extent, though union through incised adipose tissue is usually effected by means of connective tissue, which is not transformed into fat.

5. Cartilage has also a certain power of repair, but injured cartilage is usually replaced by fibrous tissue. Still, more or less rudimentary cartilage can be produced on a large scale, as seen in Enchondromata. When the repair is effected by cartilage the cells enlarge, and their nuclei and protoplasm divide, and give rise to many cells which become surrounded by capsules and by a hyaline matrix, which is probably secreted by the cells. It is also stated that cartilage can grow from periosteum, perichondrium, marrow, fibrous tissue, and epithelium. The change may be a direct one or by means of granulation tissue (Zeigler).

6. Bone.—Repair of bone is accomplished by a process similar to that of healing by first intention. Blood is effused around the ends of the fragments, and leucocytes, which escape from the injured vessels, infiltrate the periosteum, the medulla, and the surrounding tissues. After a time the inflammation subsides, and the broken ends of the bone become embedded in a mass of granulation tissue, called *callus*.

This mass of callus may be divided into three parts :—

1. That which replaces the medulla of the bone—*internal callus*.
2. That which forms a sheath on the outer surface of the ends of the fragments—*ensheathing callus*.
3. That which lies between the ends of the fragments—*intermediate or permanent callus*.

Gradually this mass of callus becomes firmer and more fibrous, and in some cases cartilaginous, a new periosteum is formed on its outer surface; and beneath this, by means of osteoblasts, ossification commences and spreads to the internal and then to the intermediate callus, thus firmly

uniting the broken fragments. After a time the internal and external ossified callus is removed, the permanent callus alone remaining to fix the fragments.

7. Muscle.—After section through muscles, reunion is generally accomplished by means of ordinary fibrous tissue. The muscular fibres degenerate, they become swollen, lose their striation and atrophy. The nuclei, however, remain. Between the cut ends of the muscle a mass of granulation tissue forms, and connective tissue corpuscles penetrate between the muscular bundles and unite them together. There is, however, good evidence that new muscle cells can be produced from the muscle nuclei. These nuclei remain after the rest of the fibres have degenerated—they enlarge and increase in number, and form elongated cells. These gradually become striated, and reproduce the muscular fibres.

8. Nerve Fibres have no great power of repair. Regeneration is accomplished by outgrowths of pre-existing nerves, but the process by which this is accomplished is as yet undecided. According to one view, however, the nuclei of the primative sheath in the two ends of the divided nerve are said to proliferate and give rise to new axis-cylinders. In any case the axis-cylinders and the primative sheaths are first formed, and then the white substance of Schwann.

Nerve Cells once destroyed are probably never replaced.

9. Blood-vessels, as we have seen when treating of inflammation, have great power of regeneration and repair. There are two stages in their production:—

(*a*) In the adult all new vessels are outgrowths of pre-existing vessels. They begin as conical-shaped solid buds of protoplasm, growing from the outer surface of capillary vessels. These buds, at their apeces, become prolonged into fine filaments of granular protoplasm, which gradually elongate and send off branches. The processes are at

first solid and may either become united with the walls of adjacent vessels, or several of them may unite together, or they may return and become attached to the original vessel, thus forming arches.

From these solid offsets other branches spring, thus forming a more or less evident net-work. Nuclei now appear amongst the granules, and the solid buds and processes become hollowed out by their central protoplasm becoming fluid, thus converting them into rudimentary vascular channels, which are connected with the pre-existing vessels. At first, the walls of these young vessels are homogeneous, but after a time protoplasm gathers round the nuclei, forming the endothelium cells of the vessel wall.

(*b*) Sometimes spindle-shaped or branching cells are seen to become connected with the offshoots of the capillary walls, and by the excavation of the central part of their protoplasm become transformed into new vessels in a manner similar to that which occurs in the vascular buds.

10. EPITHELIUM has great power of repair. Normally, all surfaces covered by epithelium have their cells constantly replaced by new cells. Even when removed from the body, epithelium lives for some time, as seen in skin grafting. It is always regenerated from pre-existing epithelium.

Glandular Epithelium has also a certain power of repair as seen in the tubules of the kidney.

II.—HYPERTROPHY.

I. DEFINITION—

HYPERTROPHY—over-nutrition—is defined as an increase in the size and number of the constituent elements of a tissue or organ; the affected parts retaining their functions and normal relations to each other. The term is restricted to cases of enlargements, where the general structure is retained.

Hyperplasia—also called *Numerical Hypertrophy*—is a term used to express increase in number of constituent elements.

Increase in size of constituent elements is called *Simple Hypertrophy*.

Hypertrophy is to be distinguished—

1. From overgrowth, due to inflammation.

2. From new growths—neoplasms.

3. From mere enlargements or distension of parts and organs, which may occur with actual diminution in size and number of constituent elements—as seen in hydrocephalus and in organs enlarged by tumours.

4. From enlargements due to degenerations and infiltrations—*e.g.*, a fatty liver may be much enlarged, but the normal tissue elements may have decreased.

III. Division—Hypertrophy may be—

1. General.—Affecting the whole body—*e.g.*, giants.

2. Local.—Affecting particular parts or organs, and is divided into—

(1) Physiological Hypertrophy.—Hypertrophy due to increased functional activity—*e.g.*, pregnant uterus, etc.

(2) Pathological Hypertrophy, where either—

(*a*) The cause is a morbid one.

(*b*) Or where the hypertrophy is not directly proportionate to the increased function.

III. Causes of Hypertrophy—

1. Increased Nutrition, though not *per se* a direct cause of hypertrophy, is, however, requisite for the process. Thus, in young animals, we may have increased growth, due to increased blood supply from paralysis of vaso-motor nerves.

2. **Increased Function** is a powerful factor in the production of hypertrophy—*e.g.*,

(1) Increase in size of muscles, from increased use.

(2) Where one organ has been destroyed, its fellow takes on its function—*Compensatory Hypertrophy.*

3. **Direct Stimulation**—

(1) Pressure, when intermittent, causes hypertrophy—seen in skin of hands when much used, etc.

(2) Irritation to a bone after an operation, causes overgrowth.

(3) Some poisons—*e.g.*, phosphorus—cause hypertrophy of bone.

IV. Nature of the Process—

The exact nature of the process which occurs in hypertrophy is not clearly understood.

The chief factor, however, in the process is an increase in the *number* of the constituent elements of the affected part. This is often called hyperplasia—numerical hypertrophy.

How far the individual tissue elements increase in size is somewhat difficult to decide; but that a marked increase of the size of the tissue elements can occur may be determined from the examination of the uterus in pregnancy, and from the heart in hypertrophy; in both which cases there is a marked increase in the size of the muscle cells of the affected organ.

The process of hypertrophy, therefore, is most likely an increase in the number of the constituent elements of the affected part, with possibly an increase in size of the individual tissue elements.

In any case the requisites for the process are an increased vascular and nutritive supply, and a certain formative power of the tissue elements.

III.—TUMOURS.

Tumours may be considered under the following headings—

1. Definition.
2. General Characters.
3. Structure.
4. Causation.
    (1) Predisposing Causes.
    (2) Exciting Causes.
    (3) Theory of Embryonic Residues.
5. Liability to Tumour Growth.
6. Life History.
    (1) Degenerations.
    (2) Growth.
        (*a*) Local.
        (*b*) Metastasis.
7. Clinical Characters.
8. Constitutional Effects.
9. Classification.

I. Definition—

"Tumours—New Growths—Neoplasia—are swellings not depending upon inflammation or hypertrophy, and showing no tendency to spontaneous cure."

A tumour, therefore, is a new growth which has the following characters—

(*a*) It occurs as a mass or as an infiltration.
(*b*) It has no function.
(*c*) It grows independently of the needs of the economy.
(*d*) It is persistent, and continues to grow after the irritation which caused it has been removed.

These several characters distinguish tumours from hypertrophy, and from new growths due to inflammation or to repair.

II. GENERAL CHARACTERS—

Tumours consist of elements similar to those of the normal tissues, but show greater exuberance of growth and greater varieties of size, shape, and arrangement of their constituent elements. They arise from pre-existing cells, and retain the group characters of the tissue from which they originate; thus, epithelial tumours retain the characters of epithelium, and even of the special form of epithelium from which they are derived.

III. STRUCTURE—All tumours consist of—

1. A supporting framework.
2. Tissue elements, such as epithelium, etc.
3. Blood-vessels, and often lymphatics and nerves.

IV. CAUSES OF TUMOUR GROWTH—

Little is known of the ætiology of tumours. Their causes may be classed as follows—

1. **Predisposing Causes.**—Hereditary transmission, and constitutional tendency—*e.g.*, moles, cancers, etc., are said to be hereditary.

2. **Exciting Causes.**—All kinds of irritation—*e.g.*,

(1) Irritation of soot causes chimney sweepers' cancer.

(2) Cancer of lower lip from constant use of the tobacco pipe.

(3) In the liver, cancers often commence at the spot where a gall-stone has lodged.

(4) Bony tumours may have their source in long-standing friction—*e.g.*, rider's bone.

(5) Direct damage—*e.g.*, a blow may be the starting point of cancer of the breast.

(6) Chemical irritants—*e.g.*, workers in petroleum are subject to a special form of cancer.

(7) Inflammation may cause tumour growth. Sarcoma often start from an old scar.

(8) Granulomata—new growths closely allied to tumours—are probably due to bacterial irritation.

In many cases of tumour growth no definite cause can be assigned; there is, however, in all probability a local weakening of the nutritive control of the affected part.

3. Theory of Embryonic Residues (Cohnheim). —This theory holds that certain parts of embryonic tissues may remain undeveloped, and lie dormant for a long period of time within a fully formed tissue. Then either from local weakening, or loss of power of resistance in the tissues, or from some special stimulus, or from damaged or altered blood supply, these embryonic residues take on an active growth, and thus give rise to tumours.

Examples:—

(1) Dermoid cysts which grow about the head and neck in the region of the branchial clefts, are said to be produced by a drawing in, at an early period of embryonic life, of skin which afterwards becomes covered over by growth of other tissues.

(2) Again, tumours tend to start at congenital spots—*e.g.*, from moles, etc.; and also at points where two different epithelial surfaces meet—*e.g.*, edges of lips, rectum, vagina, etc.

(3) Cartilaginous tumours—*e.g.*, in the parotid—are supposed to have their origin in small masses of embryonic cartilage of the pinna of the ear, which have become enclosed in the process of development of that gland.

V. Liability to Tumour Growth—

1. In Organs.—Those organs are most subject to new growths which undergo periodic evolution—*i.e.*, in which the functions are periodic—*e.g.*, the stomach, mamma, uterus.

2. In Tissues.—Connective tissues and epithelium are very liable to tumours; vascular tumours, on the other hand, are comparatively rare.

VI. Life History of Tumours—Tumours once formed, tend—

(1) To Degenerate; (2) To Grow.

1. Degenerations—

(1) Fatty degeneration—the most common.
(2) Mucoid; colloid.
(3) Calcification.
(4) Pigmentation.
(5) Inflammation, ulceration, and suppuration.
(6) *Metaplasia* is a tendency to the transformation of one type of tissue into another—thus, simple connective tissue tumours may become sarcomata; a papilloma may become an epithelioma.

2. Tendency to Growth—

(1) Tumours may be subject to periodic growth and quiescence—*e.g.*, fibroid tumours of the uterus enlarge during the menstrual period, and then diminish in size.

(2) Tumours may spread—

(*a*) Locally, or be
(*b*) Diffused through the body.

(*a*) Local Increase occurs—

(1) By pressure on the surrounding tissues.
(2) By transformation of the surrounding tissues—*e.g.*, as seen in some adenomata of the liver.
(3) By infiltration or invasion.

(*b*) Metastasis—

This term is applied to secondary tumours arising from the transportation of the elements of the original new growth to distant parts. The elements, thus carried, may consist of parts of the original tumour, or of the agent that set up the original growth. The secondary growths resemble the primary ones in their structure.

Mode of Transference—

(1) By *Arteries or Veins.*—The tumour may grow into the vessels, and there form masses, small parts of which may become detached and be transported elsewhere; or the cells of the tumour may be carried by the blood current, and give rise to secondary growths in other parts of the body. Thus, growths connected with the portal system cause secondary growths in the liver, etc.

(2) By *Lymphatics.*—In this case, either the cells of the original tumour, or some deleterious agent is carried by the lymphatics, and thus gives rise to metastasis.

It is a question whether tumour cells can migrate like white corpuscles, but it is quite possible they may do so.

Mode of Growth of Secondary Tumours. — These tumours may grow—

(1) By multiplication of the transported cells—the cells dividing, and thus giving rise to a new growth similar to the primary tumour; or

(2) By the so-called "spermatic influence."— The individual cells do not in this case proliferate, but, by local contact with the cells of the invaded tissue, they exercise an influence which causes the cells to proliferate and give rise to new growths.

The first view is the more probable one.

Multiple tumours are not all secondary, for they may have their origin in a general infection, taking place at many points at one and the same time.

VII. Clinical Characters of Tumours—

Tumours in their clinical aspect are regarded as either Innocent or Malignant.

1. **Innocent Tumours** are those which grow slowly, are homologous, resemble the tissue from which they grow, consist of fully formed tissue, and are enclosed within a capsule. They are circumscribed, do not infiltrate locally nor involve lymphatic glands, are not metastatic, and do not recur when removed.

2. **Malignant Tumours**, on the other hand, have rapid growth, are heterologous, have no capsule, infiltrate surrounding parts, and are consequently fixed and adherent. They involve lymphatic glands, are metastatic, recur when removed, and give rise to constitutional effects—cachexia.

VIII. Constitutional Effects of Tumours—

Cachexia—When the general nutrition and functions of the body become impaired, and general ill-health and exhaustion result, we have the state of cachexia. These constitutional effects are supposed to be due to absorption of the degenerated products of the new growths.

Death, as a result of tumour growth, may occur—

(1) By direct pressure on important organs, causing mechanical interference with vital functions.
(2) By Exhaustion.
(3) By Ulceration.
(4) By Hæmorrhage.

IX. CLASSIFICATION OF TUMOURS—Tumours may be classified as follows—

According to their Structure—

(1) SIMPLE TUMOURS.—Tumours of fully formed tissues.

(2) TUMOURS composed of tissues that are not fully formed—*i.e.*, are more or less EMBRYONIC in type.

(3) EPITHELIAL TUMOURS.—Tumours characterised by exuberance of growth.

(4) CYSTIC TUMOURS.

Other Classifications:—

According to clinical characters—

(*a*) Malignant.
(*b*) Non-malignant.

According to the embryonic layer to which they belong, into—

(*a*) Mesoblastic, or connective tissue tumours.
(*b*) Hypoblastic—epithelial tumours made up of cells like epithelium.
(*c*) Teratomata—tumours of hypoblastic, mesoblastic, and epiblastic tissues.

According as to whether the tumour resembles or not the tissue elements in which it grows into—

(*a*) Homologous.
(*b*) Heterologous.

NOTE.—The classification of Tumours according to their *Structure* is the one that will be followed.

TABLE.

Classification of Tumours according to their Structure:—

I. Simple Tumours—Connective Tissue Tumours.

(1) Fibroma.
(2) Myxoma.
(3) Glioma.
(4) Lipoma.
(5) Enchondroma.
(6) Osteoma.
(7) Lymphoma, etc.
(8) Angioma.
(9) Myoma.
(10) Neuroma.

II. Tumours composed of Embryonic Connective Tissue—Sarcomas.

1. Round-celled Sarcomas—
   (1) Lympho-sarcoma.
   (2) Glio-sarcoma.
   (3) Psammoma.
   (4) Alveolar Sarcoma.
   (5) Melanotic Sarcoma.

2. Spindle-celled Sarcomas—
   Fibro-sarcoma.

3. Myeloid Sarcomas.

4. Special Forms of Sarcomas—
   (1) Cylindroma.
   (2) Myxo-sarcoma.
   (3) Chondro-sarcoma.
   (4) Osteo-sarcoma.

III. **Epithelial Tumours**—New growths of Epithelium.

1. Simple Epithelial Tumours—
   (1) Papilloma.
   (2) Adenoma—Simple Adenoma.
      (*a*) Acinous forms.
      (*b*) Tubular forms.

2. Malignant Epithelial Tumours—Carcinomas.
   (1) *Epithelial Cancers*—Epitheliomas.
      (*a*) Squamous-celled.
      (*b*) Cylindrical or columnar celled—Malignant Adenoma.
   (2) *Acinous Cancers*—Cancers proper.
      (*a*) Schirrus.
      (*b*) Encephaloid.
      (*c*) Colloid.

IV. **Cystic Tumours**—Cysts.

1. Spurious Cysts.

2. True Cysts.
   (1) Cysts due to accumulations within actual or potential cavities.
      (*a*) Retention cysts.
      (*b*) Exudation cysts.
   (2) Cysts of new formation—Cystomata.
   (3) Developmental or Congenital Cysts.

I.—SIMPLE TUMOURS.

Tumours composed of the various fully formed simple tissues.

I.—FIBROMA.

I. DEFINITION—

Fibromata are connective tissue tumours consisting of connective tissue fibres and cells.

II. GENERAL CHARACTERS—

Fibromata are well defined nodules or masses resembling the connective tissue from which they grow. They may therefore be firm and tense like tendons, or soft like areolar tissue.

III. DIVISION—

1. LAMINATED FIBROMATA.—Hard flat growths, with a structure similar to that found in the cornea—lamillæ of fibrous tissue with flattened connective tissue corpuscles between the layers. They occur on the surface of the spleen, pleura, pericardium, but it is doubtful whether they are true fibromas.

2. FACICULATED FIBROMATA are divided into two classes, according to their consistence—(1) Hard; (2) Soft.

1. Hard Fibromas are pale, glistening, white tumours, varying much in size. They are firm, tough, sharply defined growths, with a definite capsule, grating when cut, and on section show wavy bundles of fibres like watered silk.

STRUCTURE. — They consist of interlacing bundles of white fibrous tissue, and have few cellular elements. They may be irregularly faciculated in the arrangement of their fibres; or laminated—arranged concentrically around the blood-vessels or nerves.

Sites—

(1) Skin, fascia, tendons.
(2) Periosteum—*e.g.*, of lower jaw (fibrous epulis).
(3) Round nerves (false neuroma).
(4) Connective tissue of glands, kidney, mamma.
(5) Uterus (uterine fibroids).
(6) Rectum (fibrous polypus).

2. Soft Fibromas are soft succulent tumours of grey translucent appearance, diffuse or multiple, pedunculated or sessile, and either with or without a capsule.

Structure.—They consist of a loose network of fibres, like areolar tissue, there being no regular arrangement of the connective tissue fibres. They have many cells, both connective tissue cells and leucocytes.

Sites—

(1) Subcutaneous, submucous tissues—*e.g.*, on the scalp, wens; molluscum fibrosum—small sessile or stalked tumours spread all over the body; on the scrotum—on the labia.
(2) Intermuscular septa, periosteum.
(3) Mamma.

IV. Clinical Characters—

1. Innocent.
2. Non-metastatic.

V. Degenerations—

1. Mucoid.
2. Fatty.
3. Calcareous.
4. Inflammation.
5. Ulceration and Softening.

VI. Transition Forms—

1. Fibro-sarcoma.
2. Fibro-lipoma.
3. Fibro-myoma, etc.

II.—MYXOMA.

(Mucous Tissue Tumours.)

I. DEFINITION—

These are new growths made up of mucoid tissue, similar to the embryonic tissue of the umbilical cord. Many new growths undergo myxomatous degeneration, and thus resemble myxomas; but true myxomata consists of mucoid tissue from the commencement.

II. GENERAL CHARACTERS—

Myxomata are soft, homogeneous, gelatinous tumours, usually enclosed within a capusle. They may be of large size, single or multiple, rounded or lobulated, and are of a yellow-grey or red-white colour. When cut into, a glairy-looking fluid oozes from the cut surface.

III. STRUCTURE—

Myxoma consists of mucoid tissue which is made up of two kinds of cells—

(1) Large angular branching cells which anastomose together; and of

(2) Spheroidal, oval, or fusiform cells.

The outlines of the cells are very indefinite; there is much intercellular substance, which presents a homogeneous, gelatinous appearance, and contains much mucin.

IV. SITES—

Myxomata occur, at the later periods of life, in—

1. Subcutaneous, subserous, and submucous connective tissue—*e.g.*, nasal polypi.
2. Intermuscular septa, periosteum.
3. Medulla of bone.
4. Connective tissue of mamma.
5. Round nerve trunks (neuromata).
6. Placenta (uterine hydatids).
7. Arachnoid and choroid plexuses of brain.

V. Clinical Characters—

1. Non-malignant.
2. Non-metastatic.

VI. Degenerations—

1. Mucous softening.
2. Colloid degeneration.
3. Hæmorrhage.

VII. Transition Forms—

Myxo-fibroma, Myxo-lipoma, etc

III.—GLIOMA.

I. Definition—

These are tumours formed of connective tissue similar to that which is found in the brain and spinal cord—neuroglia.

II. General Characters—

Gliomas vary much in appearance; they resemble the white matter of the brain, but are softer, forming more or less firm grey gelatinous-looking masses, usually of small size, solitary and imperfectly defined from the surrounding tissue, for they have no capsule. Sometimes they are of a grey-red, or even dark-red, colour, due to the number of their blood-vessels.

III. Sites—

1. Brain and Spinal Cord.
2. Retina.

1. Glioma of Brain and Spinal Cord—

1. Structure.—Gliomas of the brain and spinal cord consist of neuroglia cells—rounded, oval, nucleated cells, branching and forming network of interlacing filaments.

They are small, more or less defined growths, often very vascular, the blood-vessels being dilated and sacculated.

2. Degenerations.—Gliomas are liable to—
   (1) Fatty degeneration.
   (2) Caseation.
   (3) Softening and disintegration.

3. Clinical Characters—
   (1) Innocent.
   (2) Non-metastatic.

2. Glioma of the Retina—

1. Structure.—These tumours consist of cells similar to those of the granular layers of the retina—*i.e.*, small rounded or branched cells. They are not true gliomas, but are most likely closely related to sarcomas. (See "Sarcomas.")

2. Clinical Characters—
   (1) Recur locally after removal.
   (2) Metastatic.

IV.—LIPOMA.

I. Definition—

Lipomata — fatty tumours — are tumours composed of adipose tissue, and are one of the most common forms of new growth.

II. General Characters—

Lipomata are soft or firm, rounded, lobulated, sessile, or pedunculated masses—often of very large size—solitary, but may be multiple.

III. Structure—

They consist of lobules of fat bound together by vascular connective tissue—the individual fat globules, as well as the lobules, being larger than normal. The tumour is surrounded by a thin capsule from which it can be readily shelled out.

IV. Sites—

Fatty tumours usually occur—

1. In situations where adipose tissue normally exists and are especially common in the trunk, back, nates, etc.
2. In situations where there is no fat—*e.g.*, submucous coat of the intestine, and in the dura mater.
3. Also said to occur in the kidney, heart, bone; rare in muscle.

V. Clinical Characters—

1. Innocent—do not recur after removal.
2. Non-metastatic.

VI. Degenerations—

1. Calcification.
2. Mucoid softening.
3. Inflammation.
4. Ulceration.
5. Necrosis, Gangrene.

VII. Transition Forms—

1. Fibro-lipoma.
2. Myxo-lipoma.

V.—ENCHONDROMA—CHONDROMA.

I. Definition—

Enchondromata are tumours which consist chiefly of cartilage, though they rarely grow from pre-existing cartilage.

Ecchondrosis is a term applied to small cartilaginous masses which, both in appearance and in structure, more closely resemble normal cartilage than do true enchondromata. They are simple outgrowths of pre-existing cartilage, and are found usually in connection with costal cartilages and with the cartilages of the larynx.

II. General Characters—

Macroscopic.—Enchondromata are simple or multiple growths which vary much in size, sometimes being as much as three feet in circumference. They are usually smooth, hard, elastic, lobulated tumours, which consist either of a single mass or of several masses bound together.

Sometimes they are soft and tend to grow rapidly.

Microscopic.—Enchondromata are made up of hyaline cartilage, consisting of cells and matrix.

The *Matrix* is homogeneous, hyaline, with occasionally some white or yellow fibres present. The tumour is composed of groups or islands of hyaline cartilage, separated by septa of vascular fibrous tissue. The septa are derived from the capsule which surrounds the tumour. Sometimes both septa and vessels are wanting.

The *Cells* are similar to those found in hyaline cartilage, but are more irregular in size and shape, and may or may not have a capsule.

III. Sites—Enchondromata may grow from—

1. Connective tissue, periosteum, bone.
2. Testicle, parotid gland, mamma, — rarely in other glands.

IV. Clinical Characters—

1. Most enchondromata are innocent.
2. The softer forms, especially those that are connected with the medulla of bones, and those that grow from glands, are often malignant.

V. Degenerations—

1. Softening due to myxomatous degeneration.
2. Calcification.
3. Ossification.
4. Inflammation.
5. Ulceration.

VI. Transition Forms—

1. Myxo-chondromata. — Cartilaginous tumours, consisting of branching stellate cells similar to those in mucous tissue. They grow from the vertex and from the base of the skull.

2. Fibro-chondromata.—Cartilaginous tumours, characterised by having bundles of white fibrous tissue arranged irregularly, or in circles, around the cartilage cells.

3. Osteo-chondromata.—Growths in which the matrix, consisting of fibrous trabeculæ, is partially calcified. The cells are small, rounded, oval, or angular in shape, and have no definite capsule.

4. Chondro-sarcomata—In which the cells are spindle-shaped, and are embedded in a hyaline matrix with blood-vessels. They have no capsule and grow rapidly.

VI.—OSTEOMA.

I. Definition—

Osteomata are new growths which consist of true bone. They are often called Exostosis.

II. General Characters—

Osteomata are usually multiple tumours, and start in early life.

III. Division—

There are two varieties of osteomata—

1. Ivory Osteomata. — These are smooth rounded growths, with a broad base, and are composed of dense compact bone, similar to ordinary compact bone.

2. Spongy or Cancellous Osteomata are small, usually pedunculated growths, composed of cancellous or spongy

bone—*i.e.*, thin bony trabeculæ with large medullary spaces, and are at first covered by a layer of cartilage. They usually grow from the cancellous tissue of bone.

IV. SITES—

1. IVORY OSTEOMATA grow from—
   (1) The skull bones—internal and external table.
   (2) The upper or lower jaws.
   (3) Pelvic bones.
   (4) Scapula.

2. CANCELLOUS OSTEOMATA grow from cartilage or bone —especially near the epiphyses.

Osteomata growing from connective tissue are, in all probability, other forms of tumours which have become calcified.

V. CLINICAL CHARACTER—

True osteomata are non-malignant.

VII.—LYMPHATIC TUMOURS.

These tumours form two groups :—

(1) LYMPHANGIOMATA.—Tumours composed of lymphatic vessels.

(2) LYMPHOMATA.—Tumours having the structure of lymphatic glands.

I.—LYMPHANGIOMA.

I. DEFINITION—

Lymphangiomata are tumours composed of dilated lymphatic vessels, with atrophy of the surrounding tissues.

II. DIVISION—

1. SIMPLE LYMPHANGIOMATA—Lymphangiectasis.
2. CAVERNOUS LYMPHANGIOMATA.

III. General Characters & Structure—

1. Simple Lymphangiomata are soft elastic swellings of rare occurrence. They consist of dilated lymphatic vessels, with possibly some lymphatic vessels of new formation.

2. Cavernous Lymphangiomata are tumours which consist of enormously dilated lymphatic vessels, forming lymph spaces separated from each other by fibrous septa, and lined by a layer of endothelial cells. They sometimes form cyst-like cavities, full of coagulated lymph.

IV. Sites—Either form may be congenital or acquired.

1. Congenital—

(*a*) On the tongue—macroglossia.

(*b*) On the lips—machrochelia.

(*c*) On the skin—lymphatic nævi.

2. Acquired.—On the skin, especially on the thorax and on the thighs.

V. Degeneration—

Rupture, causing great loss of lymph.

II.—LYMPHOMA—LYMPHADENOMA.

I. Definition—

1. Lymphomata are tumours made up of lymphoid or adenoid tissue—*i.e.*, tissue similar to that found in lymphatic glands, etc. The term is also used of mere enlargements or outgrowths of lymphatic glands.

2. Lymphadenoma is a term used in several meanings—

(*a*) It is used synonymously with Lymphoma.

(*b*) It is used of multiple growths that are overgrowths —hyperplasias—of lymphoid tissue in the several situations in which that tissue occurs.

(*c*) It is used synonymously with Lympho-sarcoma.

II. General Characters—

1. Macroscopic.—Lymphomata vary much in character.

(*a*) Some are soft, greyish white tumours, containing a milky white juice. They grow rapidly, and may reach a large size. They have many cells and little stroma.

(*b*) Other forms are firmer, having fewer cells and much stroma. They are small tumours, and grow more slowly.

2. Microscopic. — Lymphomata have a structure very similar to lymphoid or adenoid tissue.

This tissue consists of a delicate reticulum of homogeneous fibres clothed with endothelial cells. At the crossing of the network, nuclei are often visible. Within the spaces of the mesh-work are lymph and lymph-corpuscles. Hence adenoid tissue has three elements :—

(*a*) A network of fibrillæ—branching cells.

(*b*) Endothelial plates.

(*c*) Lymph and lymph corpuscles.

III. Division—

Lymphomata, therefore, may be overgrowths of one or other, or all, of these elements of adenoid tissue; hence, there are four chief varieties of lymphomata :—

1. Lymphomata — made up of all these elements of adenoid tissue. They grow from lymphatic glands, and from lymphoid tissue in other situations. They have the normal arrangement of adenoid tissue.

2. Lymphomata in which there is a marked increase in the lymph corpuscles. They are called Lympho-sarcomata. They may be of large size—infiltrate locally, and are metastatic.

3. Lymphomata in which the stroma is much increased in amount. They occur in the disease known as Hodgkin's disease, in which there is an enlargement of lymphatic glands. Similar growths also occur in the liver, lungs, etc.

4. Lymphomata in which we find masses of epithelial cells enclosed in alveolar-like spaces of fibrous tissue. These tumours are difficult to distinguish from cancers.

IV. Sites—

Lymphomata occur in any of the situations in which lymphoid tissue occurs normally; hence, they grow in connection with lymphatic glands, with the mediastinal glands, etc.

V. Clinical Characters—

Lymphomata are usually innocent growths affecting individual glands or groups of glands.

The softer forms, which are rich in cells and rapid in their growth, tend to become malignant.

VIII.—ANGIOMA.

I. Definition—

Angiomata are vascular tumours, consisting of dilatations of pre-existing blood-vessels, with many vessels of new formation.

II. Division—

There are two chief varieties of angiomata—

1. Simple Angiomata.
2. Cavernous Angiomata.

III. General Characters & Structure—

1. Simple Angiomata are tumours composed of mere dilatations of blood-vessels, arteries, veins, capillaries, along with some vessels of new formation. They are usually congenital, and consist of dilated blood-vessels densely packed and bound together by bundles of delicate white fibrous tissue. The vessels may be much dilated and imperfectly formed.

(*a*) Those consisting of dilated arteries are often pulsatile growths, very vascular, and yield arterial blood when punctured.

(*b*) Those composed of veins have a blue or purple colour.

(*c*) Those composed of dilated capillaries are usually congenital and are known as nævi—mother's marks. They may be well defined or diffuse growths of a red, violet, or purple colour.

2. Cavernous Angiomata are vascular growths, usually surrounded by a dense fibrous capsule, though they may be diffuse. They are of a bluish colour, and are composed of enormously dilated blood-vessels, forming irregular-shaped branching spaces, separated from each other by thin septa of fibrous tissue, and lined by endothelial cells. They communicate with arteries or veins, especially the latter, and hence are full of blood. They may be congenital.

Angiomata of the liver deserve special notice. They are single or multiple growths, from the size of a pea to a walnut, and form dark-brown patches, not raised above the general surface of the liver. They are developed as age advances, and are never congenital. They are formed by dilatation and coalescence of the capillary blood-vessels of the lobules of the liver; the walls of the vessels giving way, thus forming large irregular spaces filled with blood. The cells become atrophied from the pressure upon them.

IV. SITES—

1. SIMPLE ANGIOMATA occur—

(1) On the skin, especially about the head and face, forming port-wine stains—mother's marks.

(2) Sometimes in the subcutaneous and submucous tissues.

2. CAVERNOUS ANGIOMATA occur—

(1) Most commonly in the liver.

(2) Rarely in subcutaneous tissues.

(3) In other organs, such as the spleen, kidney, etc.

V. CLINICAL CHARACTER—

Innocent.

VI. DEGENERATIONS—

The usual secondary changes in angiomata are hæmorrhage, thrombosis, and formation of blood cysts.

IX.—MYOMA.

I. DEFINITION—

Myomata are tumours composed either of striped or of non-striped muscular fibres.

II. DIVISION—

1. RHABDOMYOMATA—tumours of striped muscle.

2. LEIOMYOMATA—tumours of non-striped muscle.

III. GENERAL CHARACTERS—

1. Rhabdomyomata.—Tumours composed of striped muscular fibres are very rare. They are probably always partly sarcomatous in nature.

SITES—In the heart, tongue, kidney, testicle.

2. Leiomyomata.—Tumours of non-striped muscular fibres are nodulated growths, composed of bundles of non-striped muscular cells, plaited and interwoven, and surrounded by fibrous tissue, giving in section an appearance like balls of cotton.

They are somewhat difficult to distinguish from some forms of sarcomata, but are known by the oblong rod-shaped nuclei of the muscle cells, whereas in spindle celled sarcomata the nuclei are oval in shape.

SITES—

(1) In uterus—uterine fibroids.

(2) In several situations where there are non-striped muscle—*e.g.*, coats of the intestines.

IV. CLINICAL CHARACTERS—

Innocent, but a secondary growth has been found in the liver.

V. DEGENERATIONS—

1. Fatty degeneration.
2. Softening.
3. Mucoid degeneration.
4. Calcification.
5. Inflammation.
6. Hæmorrhage.

X.—NEUROMA.

I. DEFINITION—

Neuromata are tumours composed of nervous tissue—nerve fibres or nerve cells.

II. DIVISION—

1. Ganglionic neuromata.
2. True neuromata.
3. False neuromata.

III. General Characters and Sites—

1. Ganglionic Neuromata.—Tumours composed of nerve cells—extremely rare. Existence doubted.

2. True Neuromata.—Tumours composed of white or grey nerve fibres, with more or less connective tissue. They are dense, hard, white, cylindrical growths, occurring on nerve trunks or at the cut ends of nerves. They are rare.

3. False Neuromata.—Tumours not composed of nerve fibres, but of hypertrophy of the connective tissue sheaths—neurolemma of the nerve fibres, or of fatty, mucous, or fibrous tissue.

Tubercula dolorosa—the so-called painful subcutaneous tubercles—are round, exceedingly painful, subcutaneous growths, the exact nature of which is undecided, but they are probably myomas.

IV. Clinical Characters—

Neuromata are innocent growths.

II.—SARCOMA.

I. Definition—

Sarcomata are tumours composed of embryonic connective tissue—viz., connective tissue that is cellular in character, but which tends to develop into fully-formed tissue.

II. General Characters—

Sarcomata vary much in character. They start in early or middle life, are usually distinct separate tumours, and have a more or less definite capsule. By pressure, they cause the destruction of the surrounding tissues; but they also infect locally, changing neighbouring tissues into that of a sarcomatous character.

III. STRUCTURE—

Sarcomata consist of (1) Cells, (2) Stroma, and (3) Blood-vessels.

1. Cells.—The cells are usually masses of protoplasm, with no distinct cell wall—with one, or sometimes several, nuclei and nucleoli. There are three chief forms.

(1) ROUND CELLS—

(*a*) *Small* round cells, about the size of a lymph corpuscle, but having a simple round or oval nucleus. They are very granular, and have little protoplasm round the nucleus.

(*b*) The *Large* round cells have a distinct amount of protoplasm, and have a round or oval nucleus.

(2) SPINDLE CELLS.—These are elongated cells, pointed at their ends, and have a single oval nucleus and a nucleolus. They present two kinds—the large spindle and small spindle cells differing from each other in size only.

(3) MYELOID CELLS are large flat masses of protoplasm, irregular in shape, with several oval nuclei. They are like giant cells.

2. Stroma.—The intercellular substance varies much in amount but is usually scanty, and may be homogeneous, granular, or fibrillated. It surrounds individual cells, and very rarely forms alveolar spaces, as in carcinoma.

3. Vessels.—The blood-vessels are embryonic in type, having very thin walls, and ramify amongst the cells, not in a definite stroma, as in cancers. The vessels may, in fact, form mere spaces between the cells of the tumour.

IV. SITES—

Sarcomata grow from connective tissue, and occur wherever it is found. The usual situations are—

1. Skin, fascia, inter-muscular, septa, bone, periosteum.
2. Lymphatic glands, brain, ovary.
3. Rarely in liver, lungs, uterus.

V. Clinical Characters—

Sarcomata, as before stated, start in early and middle life. Their rate of growth and malignancy depend upon the special varieties. The softer forms grow most rapidly, infiltrate locally, and are metastatic; other forms, however, are by no means innocent.

Most sarcomata recur locally when removed, are metastatic, and tend to spread by the blood-vessels, not by the lymphatics, like cancers. The secondary growths have a similar structure to the primary ones, and are most liable to occur in the lungs. (For "Differences between Sarcomas and Carcinomas," see page 137.)

VI. Degenerations—

The most common secondary changes are :—

1. Softening.
2. Ulceration.
3. Mucoid, fatty, calcareous degenerations.
4. Caseation.
5. Ulceration, hæmorrhage.

VII. Classification—

1. Round-celled Sarcoma.
    - (1) Lympho-sarcoma.
    - (2) Glio-sarcoma.
    - (3) Psammoma.
    - (4) Alveolar sarcoma.
    - (5) Melanotic sacroma.
2. Spindle-celled Sarcoma.
    - Fibro-sarcoma.
3. Myeloid Sarcoma.
4. Special Forms.
    - (1) Cylindroma.
    - (2) Myxo-sarcoma.
    - (3) Chondro-sarcoma.
    - (4) Osteo-sarcoma.

I.—ROUND-CELLED SARCOMA.

There are two forms of the round-celled Sarcomas.

1. Small Round-celled Sarcomas—

1. Definition.—These are soft vascular growths, brain-like in consistence, hence called encephaloid or medullary. They grow rapidly and often reach a large size.

2. Structure—

(1) The *Cells* are very similar to leucocytes, having little protoplasm and a single oval nucleus as above described.

(2) The *Stroma* is small in amount, homogeneous or granular.

(3) The *Vessels* have very thin walls, and often consist of mere channels between the cells.

3. Sites.—They grow from fibrous tissue—hence most commonly from periosteum, bone, skin, subcutaneous tissues, and testicle.

4. Clinical Characters—

(1) Rapid growth.
(2) Malignant.

5. Degenerations—

(1) Mucoid and fatty degeneration.
(2) Ulceration.
(3) Hæmorrhage.

2. Large Round-celled Sarcomas—

1. Definition.—These tumours are somewhat firmer than the last form, and grow more slowly.

2. Structure—

(1) The *Cells* are larger, have more protoplasm, have a single large oval nucleus, but may have more than one.

(2) The *Stroma*—often as a network.

(3) The *Vessels* have very thin walls.

3. Sites.—Same as the small round-celled.

4. Clinical Characters.—Less malignant than the small-celled form, but still are metastatic.

5. Degenerations.—Similar to last form.

VARIETIES OF ROUND-CELLED SARCOMAS.

1.—LYMPHO-SARCOMA.

I. Definition—

Lympho-sarcomas are similar in structure to lymphomata. They are, however, softer, more vascular, and of a pinker colour. They are often multiple and of large size.

II. Structure—

Lympho-sarcomata consist of small nucleated cells embedded in a delicate network of branching and anastomosing cells, similar to these found in lymphoid tissue in lymphatic glands, etc.

III. Sites—

(1) Lymphatic glands.

(2) Lymphoid tissue of mucous membranes.

IV. Clinical Characters—

(1) Grow rapidly.

(2) Malignant.

2.—GLIO-SARCOMA.

I. Structure—

Glioma of the retina has a structure very similar to that found in the granular layers of the retina—viz., a groundwork of delicate fibrous tissue, with cells having very evident nuclei. (See "Glioma," page 109.)

II. Sites—

It occurs only in childhood—in the eye.

III. Clinical Characters—

Malignant, infects locally, and grows rapidly.

3.—PSAMMOMA.

I. Definition—

Psammomata, or angiolithic sarcomata, are usually small tumours, somewhat rounded in form.

II. Structure—

They consist of cells, which have a concentric arrangement around little vascular buds. Calcareous nodules, brain-sand, are often found in the centre of these growths, owing to colloid degeneration of the vascular buds, followed by calcification.

III. Sites—

The meninges and choroid plexuses of the brain.

IV. Clinical Characters—

1. Growth limited.
2. Non-malignant.

4.—ALVEOLAR SARCOMA.

I. Definition—

This is a rare form of tumour, and is very similar to cancer—consisting of cells within alveolar spaces, separated from each other by septa of fibrous tissue.

II. Structure—

1. The *Cells* are large, rounded, with a single nucleus; and are not epithelial in type, as in cancers.

2. The *Stroma*—Besides the fibrous septa, there is a little intercellular substance passing between the individual cells, and containing the blood-vessels.

III. Sites—

They grow from connective tissue, not from epithelium, and are thus distinguished from cancer. They occur in skin, bones, muscles.

IV. Clinical Characters—

They tend to recur locally, and are metastatic.

5.—MELANOTIC SARCOMA.

I. Definition—

Melanotic sarcomata are tumours characterised by having a brown or almost black colour, due to pigment in the cells and in the stroma.

N.B.—Melanotic sarcomata may be classed as round-celled or as spindle-celled sarcomata, for at one time the first type, at another the second type of cell predominates.

II. STRUCTURE—

The *Cells* may be round or oval, but are often spindle-shaped, and contain pigment in the form of granules. Sometimes, from degeneration and breaking down of the cells, the pigment may be seen in small masses. Pigment is also present in the stroma.

III. SITES—

Where there is pigment normally—hence seen in skin-moles, pia mater, eye, etc.

IV. CLINICAL CHARACTERS—

Very malignant—in fact, the most malignant of sarcomas. They spread by lymphatics, like cancers.

II.—SPINDLE-CELLED SARCOMA.

These are the most common forms of sacromata. There are two chief forms :—

1. Small Spindle-celled Sarcomas.

1. DEFINITION. — These growths are firmer than the round-celled varieties; they have a pink-white colour, and are semi-translucent on section.

2. STRUCTURE—

(1) The *Cells* are small and spindle-shaped, having usually a single nucleus.

(2) The *Stroma*—small in amount, homogeneous; and though it may be fibrillated, at first they have a capsule.

(3) The *Vessels* are wide, with cellular walls.

3. SITES.—Periosteum, fasciæ, subcutaneous tissue.

4. CLINICAL CHARACTER.—Not so rapid in growth, and not so malignant as the small-celled variety.

5. Degenerations.—They may become—

(1) Calcified.
(2) Cartilaginous.
(3) Fibrillated.
(4) Ulcerated.

2. Large Spindle-celled Sarcomata.

1. Definition. — They are softer than the last form; are pink-white in colour; have no capsule.

2. Structure—

(1) The *Cells* are large spindles, often with several nuclei and nucleoli.
(2) The *Stroma* small in amount and not fibrillated.

3. Sites—

(1) Periosteum.
(2) Intermuscular septa.
(3) Mamma.

4. Clinical Characters.—They grow rapidly, and are very malignant, being quickly disseminated.

5. Degenerations—

(1) Fatty degeneration.
(2) Hæmorrhage.
(3) Ulceration.

FIBRO-SARCOMA.

I. Definition—

This is a form of spindle-celled sarcomata. It is a firm, coarse-grained, white or yellowish-white growth.

II. Structure—

1. The *Cells* are few in number, spindle-shaped, and arranged in interlacing bundles.

2. The *Stroma* is abundant and fibrillated.

III. Sites—

1. Fascia, periosteum, subcutaneous tissues.
2. Rare in glands.

IV. Clinical Characters—

Not very malignant.

III.—MYELOID SARCOMA.

I. Definition—

Myeloid sarcomata are tumours which usually grow from bone, and may have a shell of bone over them. They often reach a large size.

II. Structure—

1. The *Cells* are irregular-shaped, flattened, multinucleated masses of protoplasm, like giant cells.

2. The rest of the growth consists chiefly of spindle cells, there being little stroma.

III. Sites—

Usually from bone—especially from the lower end of the femur, upper end of tibia, from the humerus, from the upper and lower jaws.

IV. Clinical Characters—

They are the least malignant of sarcomata. They grow rapidly, and infiltrate locally. Secondary growths have usually no myeloid cells.

IV.—SPECIAL FORMS OF SARCOMATA.

There are several special forms of sarcomata which cannot be classed under any of the foregoing headings. They are :—

1. CYLINDROMA.

This term is applied to several forms of tumour, with few common characters. Two forms will be here noted—

1. Tumours in which the sarcomatous tissue has undergone mucoid degeneration—Myxo-sarcoma. (See below.)

2. Tumours in which the blood-vessels are seen to be surrounded by a peculiar hyaline or mucoid sheath, with layers of cells arranged concentrically around the mass.

They have their source in the mucoid and hyaline degeneration of the tunica adventitia of the vessels and of the tissues surrounding them. They are rare, but occur in the brain.

2. MYXO-SARCOMA.

Myxo-sarcomata are tumours in which the tissue of which they are composed tends to become mucoid in character; hence, they consist chiefly of branching anastomosing cells.

They tend to recur locally, but have little malignancy.

3. CHONDRO-SARCOMA.

These are sarcomatous growths in which there is a tendency to the formation of cartilage. They are met with in glands, such as the parotid and testicle, and also in bone.

4. OSTEO-SARCOMA.

Tumours which vary much in size. Cells are rounded or spindle-shaped. They consist of bony trabeculæ with sarcomatous tissue in the spaces. They are like cancellous bone—having bony spicules, Haversian canals, and all characters of normal bone. They grow from bones, and are malignant.

III.—EPITHELIAL TUMOURS.

These tumours are composed of connective tissue, of blood-vessels, and of new growths of epithelium. They may either have a structure similar to that found in the papillæ of the skin, or an alveolar structure, corresponding to that found in many glands.

There are two chief forms—

1. Simple Epithelial Tumours.
2. Malignant Epithelial Tumours.

N.B.—*The former varieties, viz., the Simple Epithelial Tumours, are often classed under Simple Tissue Tumours.*

I.—SIMPLE EPITHELIAL TUMOURS.

I. Definition—

Simple epithelial tumours are new growths which originate from surfaces that are covered by epithelium—their cells being similar in type to the epithelium from which they grow. Their stroma consists of vascular connective tissue—fibrous tissue.

II. Division—

Simple epithelial tumours are divided into two groups—

1. Papillomata.
2. Simple Adenomata.

1. PAPILLOMA.

I. Definition—

Papillomata are outgrowths and overgrowths of papillæ, with a covering of epithelial cells.

II. General Characters—

Papillomata are rarely of large size. They include—

1. *Warts*, which are circumscribed outgrowths or cauliflower-like excrescences.
2. *Mucous Tubercles*, and
3. *Condylomata*, which are flattened elevations consisting of enlarged papillæ covered by moist and soft epithelium.
4. *Villous Tumours*, which consist of delicate branching growths, like the villi of the chorion.

III. Structure—

Papillomata consist of several layers of connective tissue surrounding a central blood-vessel, and covered on the surface by layers of epithelium. The epithelium, however, never penetrates the connective tissue, and in this respect papillomata are distinguished from carcinomata—cancers.

IV. Sites—

Papillomata occur—

1. As warts on the hands, genitals, larynx.
2. As mucous tubercles and condylomata on the anus, genitals, and on the mucous membrane of the mouth and throat.
3. As villous growths in the bladder, rectum, larynx.

V. Clinical Characters—

Papillomata are innocent tumours, but some tend to become malignant.

VI. Degenerations—

1. Pigmentation.
2. Ulceration.

2. SIMPLE ADENOMA.

I. Definition—

Adenomata are innocent tumours, which grow slowly from pre-existing glands, and have an alveolar structure very similar to that found in many secreting glands.

II. Division—

1. Acinous.
2. Tubular.

III. General Characters—

1. The Acinous forms consist of proliferations of gland tissue, and are composed of alveolar spaces surrounded by a stroma of connective tissue in which the blood-vessels ramify. They are lined by spheroidal epithelial cells. They differ, however, from glandular tissue, in being more complex in the arrangement of their alveoli, and in their cells being usually somewhat flattened and very various in shape.

2. The Tubular forms are papillæ-like growths, which consist of dilatations of pre-existing gland tubes; they thus resemble the tubular glands in their structure.

It is doubtful whether a basement membrane exists in either form. They are distinguished from cancers in the fact that the epithelium does not penetrate the surrounding structures.

IV. Sites—

1. The *Acinous form* occurs in—

(1) The mamma, ovary, testes, prostate.
(2) Kidney, liver, parotid, thyroid, and other glands.
(3) Skin—from its sebaceous follicles.

2. The *Tubular form* occurs in the mucous membrane of intestinal tract, especially of the rectum.

V. CLINICAL CHARACTERS—

Simple adenomata are innocent growths. The tubular form, however, often tends to become malignant, especially when it grows from the large intestine or from the stomach.

VI. DEGENERATIONS—

Simple adenomata tend to produce mixed forms of tumours—

1. From the stroma becoming fibrous—adeno-fibroma.
2. From the stroma undergoing mucoid degeneration —adeno-myxoma.
3. From the stroma becoming sarcomatous — adeno-sarcoma.
4. From the acini becoming dilated into cysts, accompanied with mucoid softening and fatty degeneration of the epithelium—adeno-cystoma.

II.—MALIGNANT EPITHELIAL TUMOURS.

(Carcinomata.)

I. DEFINITION—

Carcinomata may be defined as malignant epithelial growths, consisting of cells, epithelial in character and arranged in an alveolar stroma.

II. GENERAL CHARACTERS—

This class of tumours usually occurs as irregular wart-like growths, firm, hard, and on section friable and granular. The more rapidly growing forms are very vascular; the more chronic varieties have fewer blood-vessels.

Carcinomata have no capsule, and hence they infiltrate locally. The stroma is arranged round groups of cells, forming alveolar spaces containing many cells of very

various forms. The stroma never passes between individual cells.

The blood-vessels run in the stroma, and are never found between the cells.

The epithelium invades the surrounding tissues, penetrates lymphatics and lymphatic spaces; and hence, by this means, the carcinomata are quickly disseminated through the system.

III. Structure—

Carcinomata, therefore, consist of—

(1) Cells.
(2) Stroma.
(3) Blood-vessels—lymphatics.

1. Cells.—The cells vary much in character, but are epithelial in type, and are similar to the epithelium from which they originate. They present every variety of form—rounded, oval, pointed, polygonal. They are usually of large size and have a round or oval nucleus and nucleolus; sometimes several nuclei. There are no specific cancer cells.

2. Stroma.—The stroma varies much in amount, but consists of connective tissue, in part fibrillated, and in part cellular, containing fusiform cells and infiltrated with small round cells. It is arranged in irregular-shaped spaces—alveoli, which communicate with each other, and which contain the epithelial cells. By many these alveoli are regarded as lymphatics dilated from overgrowths of epithelial cells. As above stated, the stroma does not pass between individual cells, thus distinguishing carcinomata from sarcomata.

3. Vessels.—The blood-vessels run in the stroma, not between the cells, and may be few or abundant. The lymphatics and the alveoli communicate, and hence the characteristic mode of spread of this class of tumours.

Differences beween Carcinomata and Sarcomata :—

Carcinomata have—

(1) An alveolar vascular stroma.

(2) Multiform cells, nucleated, arranged in alveoli, the cells epithelial in type.

(3) No intercellular substance, and no blood-vessels between the individual cells. Blood-vessels being well formed.

(4) More malignant than sarcomata, and spread by lymphatics.

Sarcomata, on the other hand, have—

(1) A stroma which forms an intercellular substance, carrying the blood-vessels with it between individual cells.

(2) The cells are embryonic in type.

(3) The blood-vessels are rudimentary and in contact with the cells.

(4) They are very malignant growths, and spread through the blood-vessels.

IV. Development—

Cancers usually begin by some irritation. They are formed by excessive division of the epithelial cells of glands, etc., followed by general epithelial infiltration. Cancer cells, therefore, are produced by proliferation of epithelial cells.

The stroma is in part new connective tissue, and in part the structures in which the cancer grows.

V. Clinical Characters—

1. Infiltrate locally.
2. Grow rapidly.
3. Metastatic, being very malignant.

VI. DEGENERATIONS—

1. Fatty degeneration.
2. Colloid degeneration.
3. Mucoid degeneration.
4. Pigmentary degeneration.
5. Calcification.
6. Ossification.
7. Hæmorrhage.
8. Ulceration.

VII. DIVISION—

Carcinomata are divided into—

1. EPITHELIAL CANCERS.
   (1) Squamous-celled.
   (2) Columnar-celled—Malignant Adenoma.

2. ACINOUS CANCERS.—Cancers proper.
   (1) Schirrus.
   (2) Encephaloid.
   (3) Colloid.

1. EPITHELIAL CANCERS.

(Epitheliomata.)

I. DEFINITION—

In this group the cancer cells are either squamous or columnar in shape, but the alveolar arrangement of the stroma is not so well marked as in the following group—cancers proper.

II. DIVISION—

1. Squamous-celled—Epitheliomata.
2. Columnar-celled—Malignant Adenomata.

I.—SQUAMOUS-CELLED EPITHELIOMATA.

(Epitheliomata proper.)

I. Definition—

Squamous-celled Epitheliomata, or squamous-celled cancers are warty masses or cauliflower-like growths which tend to ulcerate early.

II. General Characters—

They are firm but more or less friable growths, warty-looking, and with a greyish-white granular surface. On section a turbid fluid can be squeezed out, along with curdy-looking, worm-shaped masses, consisting of epithelial cells which have undergone fatty degeneration.

III. Structure—

They are composed of columns of epithelial cells which grow into the surrounding tissues, and into the lymphatics and lymphatic spaces.

In many parts of the growth the epithelial cells form small groups or masses of cells—Cell Nests—which are composed of numbers of central squamous cells surrounded by concentric layers of larger cells. They can be got separately by scraping or squeezing the growth.

The stroma varies much in amount, and may be fully formed and fibrillated, or more or less embryonic in character, and infiltrated with leucocytes.

IV. Sites—

Epitheliomata are most common in old age—after forty, and occur—

(1) In the tongue, lower lip, cheeks, and gums.
(2) Vulva, scrotum, penis, and anus.
(3) In sites of old scars, etc.

V. Clinical Characters—

1. Infiltrate locally.
2. Recur when removed.
3. May spread through internal organs.

VI. Degenerations—

1. Ulceration.
2. Fatty degeneration of the epithelial cells.

II.—COLUMNAR-CELLED EPITHELIOMATA.

(Malignant Adenomata.)

I. Definition—

Columnar-celled epitheliomata—also called cylindrical carcinomata, or adenoid cancers—are tumours which are characterised by their cells being columnar or cylindrical in shape.

II. General Characters—

They are soft, nodular growths, usually starting in the epithelium of glands. They vary much in structure, but are usually papillæ-like outgrowths from surfaces covered by epithelium, or from the interior of mucous glands.

III. Structure—

They are similar in structure to simple adenoma, differing from this chiefly in being malignant. They consist of tubes lined by columnar epithelium, with a stroma of delicate connective tissue infiltrated with small round cells. The

epithelial cells retain more or less their columnar shape, and are arranged as a lining to the alveoli, though in many cases they entirely fill the spaces.

IV. SITES—

1. Most common in the rectum.
2. Other parts of the intestine—*e.g.* stomach, etc.
3. Uterus,
4. Liver.
3. Skin (rodent ulcer).

Secondary growths occur in liver, lungs, lymphatic glands.

V. CLINICAL CHARACTERS—

1. Infiltrate locally.
2. Metastatic.

VI. DEGENERATIONS—

1. Softening.
2. Ulceration.

2.—ACINOUS CANCERS.

(Cancers proper.)

I. DEFINITION—

These are cancers which grow in connection with glands only. They have a well marked alveolar arrangement of their stroma; and the epithelial cells are similar to glandular or spheroidal epithelium.

II. DIVISION—

1. Schirrus.
2. Encephaloid.
3. Colloid.

I.—SCHIRRUS—SCHIRROUS CANCER.

I. General Characters—

Schirrous cancers are hard, nodular, slowly growing tumours of moderate size, depressed in the centre, giving a creaking sound when cut; the section presenting a greyish white, semi-translucent aspect, dotted over with yellowish white spots. They tend to ulcerate.

II. Structure—

Schirrus consists of a large amount of stroma, and of few cells. The central part of the tumours is usually composed of a few atrophied cells surrounded by dense fibrous tissue, which, by contracting, cuts off the blood supply to the central cells, causing them to undergo fatty degeneration and atrophy. The cells at the margin of the growth are more numerous, and infiltrate the surrounding structures.

III. Sites—

1. The mamma—most common.
2. Œsophagus.
3. Pyloric end of stomach.
4. Liver.
5. Pancreas.
6. Prostate.

IV. Clinical Characters—

1. Infiltrate locally.
2. Metastatic.

V. Degenerations—

1. Fatty degeneration of the cells.
2. Ulceration.

II.—ENCEPHALOID CANCER.

I. General Characters—

Encephaloid or medullary cancers are large, soft, brain-like, rapidly growing tumours, which tend to ulcerate quickly.

II. Structure—

They consist of a scanty amount of stroma, the alveoli are large and contain a great number of cells, many of which have undergone fatty degeneration. They are very vascular growths, and there is a liability to hæmorrhage into their substance. On section they have a greyish-white, cream-coloured appearance, with here and there blotches of blood. The centre of the growth is often almost semi-fluid from the fatty degeneration and disintegration of the cells.

III. Sites—

1. Testes.
2. Liver.
3. Bladder.
4. Kidney.
5. Ovary.
6. Mamma.

IV. Clinical Characters—

1. Infiltrate locally.
2. Metastatic.

V. Degenerations—

1. Ulceration.
2. Hæmorrhage.

III.—COLLOID CANCER.

I. General Characters—

Colloid cancers, also called alveolar cancers, are clear, glistening, semi-translucent, brownish-coloured, jelly-like, or glue-like tumours, which often reach a very large size.

II. Structure—

Colloid cancer consists of a definite stroma of fibrous tissue with a more or less laminated arrangement, and with colloid matter between some of the layers. The alveolar character of the stroma is well defined—the alveoli being large and rounded and filled with colloid matter, or with epithelial cells which are undergoing colloid degeneration.

There are two views as to the mode of origin of these growths. By some they are said to be due to colloid or mucoid degeneration of the schirrous or encephaloid variety of cancer; the degenerative change commencing in the epithelial cells which enlarge and become distended with jelly-like material. According to another view they are regarded as independent growths, being considered as a distinct variety of cancer, and not as a mere degenerative form of other tumours.

III. Sites—

1. Stomach, intestine.
2. Omentum.
3. Ovary.
4. Occasionally in the breast.

IV. Clinical Characters—

1. Colloid cancers infect locally.
2. Are metastatic, but still not very malignant.

V. Degenerations—

They may soften and give rise to cysts or abscesses.

IV.—CYSTIC TUMOURS—CYSTS.

I. DEFINITION—

Cysts may be defined as more or less spherical spaces, or abnormal, closed cavities, which have fluid or semi-fluid contents and a well-defined wall or capsule.

II. GENERAL CHARACTERS—

Cysts vary much in their characters, and many different structures, having little in common, pass under this name. Most cysts, however, agree in having—

1. A WALL, composed of fibrous tissue, or of the tissue of the part in which the cysts occur.

2. A LINING, generally consisting of epithelium derived from that of the part in which the cyst grows. The epithelium, however, tends to become flattened or squamous in type, or it may take on an active growth and form several layers or papillary projections inside the cyst.

3. The CONTENTS of cysts are exceedingly various, being—

(1) Fluid or semi-fluid.
(2) Serous, mucoid, colloid, gelatinous.
(3) Blood, milk, bile.
(4) Fat, masses of cells, or other products.

III. DEGENERATIONS—Cysts are liable to undergo—

1. Fatty degeneration of the contents, with formation of cholesterine.
2. Calcification of the walls and contents.
3. Colloid degeneration.
4. Inflammation and suppuration.

IV. DIVISION—Cysts may be divided—

1. According to their *Contents*, into—
   - (1) Serous Cysts.
   - (2) Blood Cysts.
   - (3) Mucous Cysts.
   - (4) Synovial Cysts.

2. According to their *Structure*, into—
   - (1) SIMPLE — UNILOCULAR CYSTS, in which there is but a single cavity.
   - (2) MULTILOCULAR CYSTS, containing several spaces more or less separated from each other by fibrous septa, which grow out from their walls.
   - (3) MULTIPLE CYSTS—where there are several distinct simple cysts united together.
   - (4) PROLIFEROUS CYSTS — those in which there is a new growth of cysts formed by a process of budding from the primary cyst. They are sometimes called compound cysts — *e.g.*, compound ovarian cysts.

3. According to their *Mode of Origin*, into—
   - (1) SPURIOUS CYSTS.
   - (2) TRUE CYSTS—
     - (*a*) Cysts due to the dilatation of actual or potential cavities—
       - (1) Retention Cysts.
       - (2) Exudation Cysts.
     - (*b*) CYSTOMATA—Cysts of NEW FORMATION—Cysts proper.
     - (*c*) CONGENITAL or DEVELOPMENTAL CYSTS—Cysts due to errors in development—
       - (1) Imperfect closure of ducts or foramina.
       - (2) Accidental mixture of fœtal structures.

I.—SPURIOUS CYSTS.

These are often called false cysts, and owe their origin to—

1. Softening, or hæmorrhage into cavities or solid organs, the softened tissue or blood being absorbed and leaving a space surrounded by a more or less definite wall.

Examples—

(1) Cysts formed by mucoid softening in tumours, such as enchondromata and sarcomata, forming cystic sarcomata, etc., in which there are spaces full of serous, mucoid, or blood-like fluid.

(2) Cysts formed in solid organs like the brain, from necrosis of the nerve structure, owing to stoppage of the blood supply by emboli, etc.

(3) Cysts due to hæmorrhage into solid organs—*e.g.*, the brain—and the subsequent absorption of the clot, leaving distinct spaces, or cysts, called apoplectic cysts.

(4) Hæmatomata—blood cysts—local extravasations of blood—may be due to hæmorrhage into serous cavities, or into connective tissue spaces.

2. Cysts may also be formed by adenomatous growths occurring side by side with sarcomata, etc.

3. *Parasitic Cysts* by some are regarded as true cysts. They have their origin in irritation set up by animal parasites, and have a distinct wall or capsule of fibrous tissue. (See Animal Parasites.)

II.—TRUE CYSTS.

(*a*) CYSTS formed by the DILATATION of actual or potential cavities—

1. RETENTION CYSTS.
2. EXUDATION CYSTS.

1. Retention Cysts.—These cysts have thick fibrous walls, and are lined by epithelium. They owe their origin to the retention of the secretion of glands from obstruction to, and subsequent dilatation of—

(1) Excretory ducts of glands.
(2) Ultimate gland ascini.

Examples—

1. Obstruction of Excretory Ducts—
   (1) Obstruction of the ureters causes dilatation of the pelvis and calyces of the kidneys, changing the entire organ into a cyst—hydronephrosis.
   (2) Obstruction of the cystic duct causes dilatation of gall bladder; of the bile ducts, cysts in the liver.
   (3) Closure of the mouth of the vermiform appendix results in the formation of a cyst.
   (4) Dilatation of obstructed Fallopian tube forms hydrosalpinx.
   (5) Obstruction and closure of duct of parotid gland causes one form of ranula.

2. Obstruction of Ultimate Gland Ascini—
   (1) *Atheromatous* cysts—sebaceous cysts caused by the closure of the orifices of sebaceous glands. They are met with in the skin, especially in the scalp, forming wens. They have a distinct wall of fibrous tissue, a lining of epidermis, and may contain fat, epithelial scales, clolesterine, etc.

(2) *Mucous* cysts—these are owing to the closure of orifices of mucous glands. They occur in those situations in which we find mucous glands—*e.g.*,

(*a*) In nose, larynx, œsophagus, etc.

(*b*) Cowper's glands.

(*c*) Glands of Bartolin—of Naboth,—Ovula Nabothi.

Retention cysts also occur in the mamma, liver, testes, kidney.

Some cysts of the kidney, however, are congenital; others are due to chronic inflammation.

2. Exudation Cysts.—These cysts are formed by accumulation of fluid in spaces which have no ducts or orifices.

Examples—

(1) Cystic göitre—bronchocele.

(2) False bursæ—bunions.

(3) Meningocele—protrusions of the membranes of the brain or spinal cord.

(4) Cysts of the broad ligaments due to dilatation of Graafian follicles.

(5) Ganglia—These have thick walls, a laminated structure, and often contain melon-seed-like bodies, either free or attached to the walls of the cyst by a slender stalk. They may contain a clear serous or thick glairy fluid.

(6) Cysts may also arise from exudation into lymph or connective tissue spaces—often called serous cysts.

(*b*) **Cystomata**—Cysts of New Formation.—This is the only group of cysts which can be considered as new

growths—*i.e.*, growths in which there is a formation of new cysts. They correspond closely to the adenomata, and are often described as compound proliferous cysts, and include the large complex cysts met with in the ovaries—"Compound proliferous ovarian cystic tumours."

These cystomata of the ovaries may be formed in two modes—

1. By dilatation of spaces in the gland structure, thus giving rise to numerous pouches or cavities lined by columnar epithelium. These cysts may reach a large size, and are composed of a number of smaller cysts containing a semi-gelatinous material, or a watery or serous-like fluid. The walls of the cysts are thick, and in the tissue between the sacs are numerous closed follicles, derived from Pflüger's tubes, lined by columnar epithelium. By dilatation of these spaces, new cysts are constantly formed; hence the name "proliferous cysts."

2. The second mode by which compound ovarian cysts are developed, is by a process of endogenous budding—papillary processes growing out from the wall of the cyst into its interior. These processes may unite with their fellows, or pouches may form within them, thus giving rise to growths of great complexity, consisting of many secondary cysts united together by a fibrous or cellular matrix.

Compound ovarian cysts, therefore, have the following characters :—

(*a*) They are of great size and complexity.

(*b*) They have a distinct capsule, are lobulated, and are lined by columnar epithelium, often partly ciliated.

(*c*) They contain a clear fluid, or a colloid, mucoid, or jelly-like substance.

(*c*) Congenital Cysts — Developmental Cysts.—These cysts owe their origin to errors in development, and form two groups—

1. Cysts formed by the dilatation of imperfectly closed ducts or foramina—*e.g.*,

(1) Hydrocele of spermatic cord, due to imperfect closure of the funicular process of the tunica vaginalis.

(2) Allantoic cyst, from incomplete closure of urachus.

(3) Hydrorachis—dropsy of the spinal cord—associated with spina bifida. It corresponds with hydrocephalus.

2. Cysts due to accidental mixture of fœtal structures—either to a simple mixture of the layers of the embryo, as enclosure of epiblast within the mesoblast, or to a mixture of other parts of embryo.

Thus we have—

(1) Atheromatous Cysts.
(2) Cystic Kidney.
(3) Hygromata.
(4) Dermoid Cysts.
(5) Teratomata.

(1) Atheromatous Cysts.—These cysts, when placed in the subcutaneous tissues, are probably due to an involution of the epiblast to form hair follicles. They are closely allied to dermoid cysts.

(2) Cysts in the Kidney may be due to the accidental mixture of the meso- and meta- nephros.

(3) Hygromata. — The nature of these growths is not yet understood, but they may be regarded as persistent portions of the branchial clefts.

(4) Dermoid Cysts.—The most simple form of this group of cysts is seen in the skin, and is most liable to occur in the neighbourhood of congenital clefts —*e.g.*, at the outer angle of the eye, in the neck, etc. They are composed of spaces containing skin, hair, nail, sebaceous glands, etc.

(5) Teratomata — Cystic Teratoid Tumours— are growths formed by an undeveloped fœtus becoming adherent to, or enclosed within, a more fully developed one. The former kind occur most commonly in the middle line of the dorsal aspect of the body; the latter are especially met with in connection with the female genital organs. These teratoid cysts contain many different structures— not only skin, hair, etc., but also cartilage, teeth, nerve tissues, glandular structures, and other parts of an undeveloped fœtus.

Section IV.

PARASITES AND PARASITIC DISEASES.

I. Animal Parasites—

1. Entozoa.

(1) Protozoa.

(2) Vermes.

2. Epizoa.

(1) Arachnida.

(2) Insecta.

II. Vegetable Parasites—

(1) Schizomycetes—Bacteria.

(2) Blastomycetes—Yeasts.

(3) Hyphomycetes—Moulds.

Section IV.

PARASITES AND PARASITIC DISEASES.

I. Definition—

Parasites are low forms of organisms—animals or vegetables—which infect other animals or plants, lodging upon them or within them, and deriving their nourishment from the tissues and juices of their host.

II. Division—

Parasites are divided into two great classes—

1. Animal Parasites.
2. Vegetable Parasites.

I.—ANIMAL PARASITES.

I.—General Characters—

Animal parasites belong to the lowest types of the animal kingdom and are characterised by the greater simplicity of their structure as compared with that of the allied non-parasitic forms.

II. Life History—

1. Metamorphosis.—Most animal parasites exist in two or more forms; the immature, larval, or embryonic form, and the mature form. Many of them undergo a series of changes — alternation of generation — before reaching maturity; first, by sexual union producing ova which give rise to embryoes or larva, which ultimately develop into adult forms, or by a process of budding, produce colonies of the mature organisms.

2. Host.—The animal infected by the parasite is called the host.

3. Intermediate Host is the organism in which the immature forms are lodged.

4. Habitat.—The part of the body of the host in which the parasite, or its immature form, takes up its abode.

III. Effects—

The effects produced by animal parasites are very various, some, as liver-flukes in sheep, are serious; others are local manifestations of slight import.

IV. Division—

Animal Parasites are divisible into two groups—

1. Entozoa—Internal Parasites.
2. Epizoa—External Parasites.

TABLE.

1. Entozoa—Internal Parasites.

I. PROTOZOA.

(1) Amœba.
(2) Coccidium.
(3) Infusoria.

II. VERMES.

1. Trematoda.
   (1) Distoma hepaticum.
   (2) Distoma lanceolatum.
   (3) Distoma hæmatobium.
2. Cestoda.
   (1) Tæniada.
      (*a*) Tænia solium.
      (*b*) Tænia mediocanellata.
      (*c*) Tænia echinococcus.
   (2) Bothriocephalidæ.
      Bothriocephalus latus.

3. Nematoda.

(1) Trichina spiralis.
(2) Filaria sanguinis hominis.
(3) Filaria medinensis.
(4) Dochmius duodenalis.
(5) Ascaris lumbricoides.
(6) Ascaris mystax.
(7) Eustrongylus gigas.
(8) Trichocephalus dispar.
(9) Oxyuris vermicularis.

2. Epizoa — External Parasites belong to the class Arthropoda.

1. Arachnida.

(1) Acarus scabiei.
(2) Demodex folliculorum.
(3) Pentastomum denticulatum.
(4) Leptus antumnalis.

2. Insecta.

(1) Pediculus capitis.
(2) Pediculus vestimentorum.
(3) Pediculus pubis.
(4) Pulex irritans.
(5) Pulex penetrans.
(6) Cimex lectularius.

I. Entozoa. — Internal Parasites — inhabit the interior of the body of their host.

I.—PROTOZOA.

These are an unimportant group of parasites as regards man.

1. Amœba.—Only one species—Amœba Coli—has been observed in the human subject. It accompanies some forms of dysentery.

2. Coccidium found in the liver and intestines, but causes no definite symptoms.

3. Infusoria occur in the blood of some animals. They are also found in the intestines in some forms of diarrhœa.

II.—VERMES—SCOLECIDA.

The Vermes or Worms are the chief class of Animal Parasites which infect man. They belong to the three following orders—

1. Trematoda.
2. Cestoda.
3. Nematoda.

I.—TREMATODA.

(Τρεμα—a pore.)

Synonyms—Flukes, Flat-worms, Suctorial-worms.

I. General Characters—

1. Shape.—The trematoda are soft, broad, oval or leaf-shaped, flattened, unsegmented worms, with a chitinous cuticle, but with a non-ciliated ectoderm.

2. Size.—They are of small size, the largest being about three inches long.

3. Structure.—The trematodes have on their ventral aspect two or more suctorial discs, sometimes armed with chitinous hooklets, and there are often chitinous setæ on other parts of the body. The alimentary canal usually consists of a bifid tube with a distinct oral, but with no anal aperture; the mouth is terminal and placed in the centre of a muscular sucker. A well developed water vascular system is always present, consisting of a contractile sac, and of longitudinal tubes connected by transverse branches. It is an excretory apparatus. The muscular

system is well marked, but a nervous system is not always present.

Most of these worms are hermaphrodite, but in one form—Distoma Hæmatobium—the sexes are distinct.

II. Life History—

Some of the trematode worms are developed directly, but the majority undergo alternation of generation—existing in one or more forms before reaching maturity. Thus we have—

1. The Ova, which give rise to free-swimming ciliated embryoes.
2. The Sporo-cysts, formed by the free-swimming embryoes becoming encysted in the body of the intermediate host.
3. The Rediæ, the intermediate larval forms generated within the sporo-cysts, and containing—
4. The Cercariæ, the tailed larva liberated by the bursting of the rediæ. These tailed larva become encysted on grass and other plants, and thus gain entrance to the body of their host, in which they develop into the adult worm.

III. Division—

The several species of trematoda which infect man are—

1. Distoma hepaticum.
2. Distoma lanceolatum.
3. Distoma hæmatobium.

I.—DISTOMA HEPATICUM.

Synonyms—Liver Fluke, Common Fluke, Fasciola Hepatica, Distoma Fasciola.

I. General Characters—

1. Shape.—The Liver Fluke is a flat, oval-shaped, rosy or yellow tinted worm.

2. Size.—About ½ inch to 1 inch in length; ½ inch broad.

3. Structure.—The head is beak-shaped, with a small suctorial disc, in the centre of which is the orifice of the mouth. Behind this is a second suctorial disc, and between these two discs is situated the genital orifice. The alimentary canal consists of a tube which bifurcates just behind the mouth, the bifurcations again branching in a tree-like manner. They end blindly, there being no anal orifice.

Ova.—Conical in shape, about $\frac{1}{300}$ inch in diameter.

II. Life History—

The ova—formed by sexual union—are liberated from the body of the adult trematode, and give rise to the free swimming embryoes. These find their way into the intermediate host—some species of fresh-water snail—and there form sporocysts. From these cysts the rediæ escape and make their way to the liver, and there give origin to the tailed larvæ—cercariæ—which leave the rediæ when it bursts, and, passing out of the body of the snail, become encysted on the grass, etc., of moist ground. They thus gain entrance into the intestine of the sheep, and, making their way to the liver, become fully developed liver-flukes. The sheep dies, the body rots, and the ova are set free from the mature worm, to again begin the cycle of development.

Intermediate Host.—Fresh-water snails—Lymnæa truncatula.

Host.—Sheep; rare in man.

Habitat.—The liver and bile ducts.

III. Effects—

Sheep rot—Chirrosis and atrophy of the liver.

II.—DISTOMA LANCEOLATUM.

SYNONYMS—Distoma Hepaticum, Dicrocœlium, Fasciola, Planaria.

I. GENERAL CHARACTERS—

1. SHAPE.—Lancet-shaped, hence the name.

2. SIZE.—Similar to the last form, but smaller in size, being ⅜ inch to ⅓ inch long.

3. STRUCTURE.—The head does not project. There are two lobulated testes below the posterior suctorial disc, and in front of this are the uterus and ovaries.

Ova.—Exceedingly small; ·04 mm.

II. LIFE HISTORY—Not known.

INTERMEDIATE HOST.—Probably a fresh-water snail.

HOST.—Sheep, cattle; very rare in man.

HABITAT.—The bile ducts.

III. EFFECT—Little; no grave symptoms.

III.—DISTOMA HÆMATOBIUM.

SYNONYMS—Bilharzia Hæmatobia, Thecosoma, Schistosoma, Gynæcophorus.

I. GENERAL CHARACTERS—

They are dicœcious, the sexes being distinct.

1. Male—

(1) SIZE.—About ½ inch long.

(2) SHAPE.—Cylindrical, with a canal or groove—gynæphoric canal—at the posterior end of the body, in which the female is lodged.

2. Female—

(1) Size.—$\frac{4}{5}$ inch long.

(2) Shape.—Thread-like.

Both male and female have two ventral suckers, and the reproductive orifice is below the ventral sucker.

Ova.—Oval in form, $\frac{1}{180}$ to $\frac{1}{160}$ inch in diameter, with a spine at the ends or at the sides of each egg.

II. Life History—Not known.

Intermediate Host.—Not known.

Host.—Man and monkey; got by drinking water.

Habitat. — The blood. Is especially found in the inferior vena cava and portal veins, and in the vesical and hæmorrhoidal veins.

III. Effects—

The sexes unite in the blood—the ova are discharged, and pass through the walls of the bladder and ureter by means of the ulcerated surfaces caused by the parents. If in large numbers, they give rise to inflammation and hæmorrhage from the affected mucous membrane, causing endemic hæmaturia—or if the large intestine be affected, a special form of diarrhœa is the result. The ova pass out of the body by these channels, and can be found in the urine of the patient. If placed in warm water, the ova give rise to the free ciliated embryoes.

IV. Distribution—

This parasite is rarely met with in England, but is common in Egypt, Cape, Natal, also in Brazil and Mauritius.

II.—CESTODA.

(Κεστος—a girdle.)

The Cestoda are all endoparasitic worms and infect the intestinal canal of vertebrata.

I. General Characters—

The Cestodes differ from the Trematodes in being multiple in character. The tape-worm is not a single individual, but a multitude of organisms arranged in a chain, thus forming a compound jointed colony.

1. Shape.—The Cestoda are compound, flat, parasitic worms.

2. Size.—Varies much; some forms measure ¼ of an inch, others 24 feet in length.

3. Structure.—The adult worm or Strobilus consists of a number of complete sexual individuals arranged in a chain. We have—

(1) The *Head* or *Nurse*, which is usually small in size, pyriform in shape and has one or two suckers surrounded by a ring of chitinous hooklets, to enable the worm to cling to the intestines of its host. It has neither an alimentary system nor sexual organs.

(2) The *Proglottides.*—These are a series of segments produced one behind the other by a process of budding from the head or nurse. Each segment or proglottis resembles its neighbours except in size and degree of maturity. The segments furthest from the head are the oldest, the largest, and most mature; the segments next the head being immature and having no sexual organs. In an ordinary tape-worm there may be as many as 1200 of these segments.

Each proglottis has a complete water-vascular system, composed of parallel canals running on each side of the body, and united at the hinder end of each segment by

cross branches. The proglottides have no digestive organs of any kind, being nourished by imbibition. They are hermaphrodite, and produce ova by sexual union with the proglottides of other cestodes. A single proglottis may contain as many as 35,000 eggs.

II. Life History—

As above stated, the ova of the Cestoda are produced in the proglottides, which when ripe break off from the rest of the chain and are cast out of the body of their host. Within these ripe proglottides the ova are already partially developed, and when ejected are full of active embryoes. These embryoes are enclosed in a membrane to protect them from injury, and consist of a head furnished with three pairs of silicious spines or hooklets. By the decomposition of the proglottides the embryo-bearing ova are set free, reach water, and thence find their way into the stomach and intestines of their host.

The membrane enclosing the embryo is now ruptured mechanically, or digested by the gastric juice, and the embryoes are liberated. They are called PROSCOLEXES (*scolex*, a worm), and consist of a small vesicle with three pairs of silicious spines. By means of these hooklets the proscolex fastens itself to the intestinal wall, bores through it, and makes its way to the liver or other organ of its host. Here it becomes encysted, loses its hooks, and from its hinder end develops a small vesicle full of fluid.

It is now called a SCOLEX which in some tæniada are known as *hydatids*, in others as *cysticercus*. When thus encysted, the scolex is composed of a vesicle united by a narrow neck to a head similar to that of the adult tape-worm, being armed with a circlet of hooklets, and having four oscula or suckers. It has no reproductive system, nor, in fact, organs of any kind, and can undergo no further development unless it gains entrance into the intestinal canal of man or other host. This is effected by an animal eating

flesh, etc., containing the scolices, when the cysts are digested and the scolices set free. They at once lose their caudal vesicle, attach themselves to the intestinal wall of their host but do not perforate it, and in this situation soon become the head of the future tape-worm and begin to produce proglottides which again pass through the cycle of development above described. Thus we have—

1. The *Ova* discharged from the ripe proglottis.

2. The *Proscolex.*—The minute embryo liberated from the ova when taken up from water, etc., by some animal.

3. The *Scolex.*—The more advanced, but still sexually immature, embryo into which the proscolex develops when it has become encysted in the tissues.

4. The *Strobilus* or adult tape-worm, infecting the alimentary canal of its host, and composed of a head, neck, and proglottides.

III. Division—

The Cestodes which infest man are—

I. Tæniada—
1. Tænia solium.
2. Tænia mediocanellata.
3. Tænia echinococcus.

II. Bothriocephalida—
Bothriocephalus latus.

I.—TÆNIA SOLIUM.

Synonyms—Tænia cucurbitina, Tænia humana armata, Tænia lata, Tænia vulgaris.

Larva—Simple scolex, Measle, Cysticercus cellulosæ.

I. General Characters—

1. Size.—The adult worm or strobilus measures about 2 to 10 feet long.

2. Shape.—It has a small head, long narrow neck, and transversely segmented body.

3. Structure—

The *Head* is small, rounded, and about the size of a pin-head, and consists of a rostellum or beak with twenty-six hooklets, and of a wider part on which are four suckers.

Body.—Next the head comes a long, narrow, thread-like neck, followed by a series of larger segments—the proglottides. At first the segments are broader than they are long, and are immature—the remaining segments are the reverse, longer than broad, and are sexually mature proglottides. These proglottides are hermaphrodite, the genital orifices being placed alternately on each side of the body, and the male and female organs open by this common genital pore. They have a complete water-vascular system.

The *Ovary* consists of a central stem, with a number of lateral branches, each of which again branches. The testes appear as clear, white, convoluted tubes, with vesicles.

Ova are nearly spherical in shape, about $\frac{1}{150}$ inch in diameter, and are surrounded by a dense capsule which encloses the partly-developed, six-hooked embryoes. These embryoes give rise to the scolices in the flesh of the pig, the scolex in this case being called a "measle," or cysticercus cellulosæ, or bladder-worm.

II. **Life History**—As above described.

Intermediate Host.—The pig, which gets the embryoes from water—the scolices forming the "measles" of pork. Within these measles or cysts, the hooklets, which do not

decompose, are often found after the scolex has perished. They are short, broad, hook-shaped bodies, with a small knob at their base.

HOST.—Man; owing to eating imperfectly cooked, measly pork. It infects man, not only as the mature worm, but as cysticerci. The adult worm is found in man only.

HABITAT.—The immature worm is found in subcutaneous tissues, muscle, brain, eye, liver, of the pig; the mature form, in the small intestine of man.

III. EFFECTS—

Segments are found in the fæces. There is pain in belly, thirst, itching of nose and anus.

II.—TÆNIA MEDIOCANELLATA.

SYNONYMS—Tænia saginata, Tænia dentata, Tænia inermis, Tæniorphynchus, Beef tape-worm.

LARVA—Cysticercus bovis.

I. GENERAL CHARACTERS—

1. SIZE.—Larger than tænia solium, both in length and breadth, often measuring from 14 to 24 feet.

2. STRUCTURE—

The *Head* has four suckers, but no rostellum nor hooklets. Following the head is a narrow neck, and then the several segments or proglottides.

The *Ovaries* consist of many lateral processes, but these do not, as in the case of tænia solium, again branch, a character by which the proglottis of the one can be distinguished from that of the other.

Ova—Similar to those of tænia solium.

II. Life History—

Similar to that of Tænia Solium.

Intermediate Host.—Cattle.

Host.—Man.

Habitat.—Immature form, in the muscles of cattle; as many as 300 having been found in a pound of flesh taken from the psoas muscles. The mature forms occur in the intestine of man.

III.—TÆNIA ECHINOCOCCUS.

Synonym—Hydatid cyst.

I. General Characters—

1. Size.—Tænia echinococcus is a small worm, about 1/8 to 1/4 inch long.

2. Structure.—This worm consists of only four segments, including the head.

The *Head* is pointed, has four suckers and a double circlet of hooks. These hooks are about 30 to 40 in number, and are shaped like those of tænia solium, but are much smaller.

The last proglottis, when mature, is equal in size to the rest of the body, and contains the reproductive organs. The genital pores are placed on the lateral aspect of the body. The ovaries are complicated, and the ova are small but exceedingly numerous, and in them are developed the six-hooked embryoes.

II. Life History—

When ripe the proglottides drop off, pass out of the body of the host—the dog or wolf. The embryoes are now liberated on the ground, on plants, or in water, and thus

gain access to the stomach of man. They then perforate the intestinal walls, and getting into the circulation, are by this or other means carried to the liver or other organ, where they become encysted and develop a spherical vesicle which may reach a great size. They are now called HYDATIDS.

Hydatid Cysts—

1. STRUCTURE.—These cysts when fully formed are composed of three parts—

(1) The false cyst formed by the tissues of the part.

(2) The ectocyst—an opaque, gelatinous membrane of great thickness, white in colour, smooth, glistening, and laminated.

(3) The endocyst—a more opaque, granular layer, composed of nucleated cells, and covered by small white spots—brood capsules.

(4) Inside the cyst there is a colourless watery fluid which contains salts, but no albumen, a point of value in diagnosis.

Hydatid cysts are, moreover, much larger than those of cysticercus cellulosa.

2. DEVELOPMENT.—Within these cysts the scolices—echinococcus heads—are developed in the following manner:—

The inner wall of the cyst forms small vesicles—called *brood capsules*—which project into the cavity of the cyst. From the walls of these brood capsules small cup-like buds or hollows are formed, each of which gradually elongates and becomes a cæcum with its cavity opening outwards—*i.e.*, it communicates with the cavity of the brood capsule. Within these depressions or hollow buds the echinococcus head is developed, and, when mature, turns itself inside out—*i.e.*, everts itself, so that the head now projects into the brood capsule. These heads are similar to those of the adult worm, having a double circlet of hooklets and four suckers.

Development cannot proceed further than this in the human body, but if the cysts gain access to the dog, etc., then the adult tape-worm is formed in the intestine.

Secondary Cysts are often found in connection with the primary cyst. This may occur in one of three modes—

(*a*) By a process of budding out of the wall of the ectocyst, thus giving rise to a number of daughter-cysts, side by side—*endogenous cysts.*

(*b*) Again, the daughter-cysts may be formed inside the primary cyst—these are called *endogenous cysts.*

(*c*) Or, the cysts may be *multi-locular—i.e.*, composed of many separate alveoli divided from each other by dense fibrous tissue. They occur as hard, firm tumours in the liver.

Intermediate Host. — The cystic form is alone found in man.

Host.—The adult worm in the dog and wolf.

Habitat.—The *cystic* form is found in the liver, lungs, brain, heart, muscle; the *mature* form, in the intestine.

BOTHRIOCEPHALUS LATUS.

Synonyms—Broad tape-worm, Tænia lata, Tænia grisea, Dibothrium latum.

I. General Characters—

1. Size.— The largest known human tape-worm. It measures from 16 to 25 feet long, and about 1 inch broad, and consists of three or four thousand segments.

2. Structure—

The *Head* is small, oval or club-shaped, with a longitudinal groove or slit on each side. It has neither proboscis, nor suckers, nor hooklets.

The *Proglottides* are about 4000 in number, the largest being in the middle of the chain. They are each bisexual. The uterus consists of a simple, coiled-up tube, and the genital orifices are placed along the middle line of the ventral aspect—not on the sides, as in the last group.

The *Ova* are oval in shape, and about $\frac{1}{350}$ of an inch long. They have an operculum and a brown-coloured shell.

II. Life History—

The ova are set free in the body of the host, and on reaching water are there hatched. The proglottides themselves are not discharged from the intestine as is the case with tænia solium. The embryo, which has a ciliated envelope, swims about in the water till the envelope bursts and liberates six-hooked embryoes. These make their way into the muscles of some fresh water fish, and there develop into the asexual larval worm.

If the fish be eaten by man, the larva develops into the sexual form above described.

Intermediate Host.—Probably certain fish, as pike, turbot.

Host.—Man, dog.

Habitat.—Intestinal canal.

III. Effects—

Intestinal catarrh in children, but no serious effects in the adult.

IV. Distribution—

Germany, Russia, Poland, Sweden, Holland, Belgium, Ireland, England, France.

III.—NEMATODA.

(νεμα, thread—ειδος, form.)

SYNONYM — Thread worms.

I. GENERAL CHARACTERS—

The thread worms are a very large and well known group of helminths. They are simple, not compound, and do not form colonies. They closely resemble the common earth-worm, being round and thread-like without segmentation or appendages. They undergo no metamorphosis, the sexes are distinct, and there is a marked difference between the male and female—the male being smaller than the female.

STRUCTURE.—The Nematoda have a distinct alimentary canal with a mouth furnished with soft horny lips, an œsophagus, stomach, intestine, and anus. There is a thick elastic ectoderm or cuticle and a well-developed muscular system. The genital pore placed on the ventral aspect is, in the female, situated about the middle of its length; in the male, near the anus, where there is a chitinous prehensile investment.

II. LIFE HISTORY—

The life history is somewhat different in each species, and will be discussed under each.

III. DIVISION—The most common Nematoda are—

1. Trichina spiralis.
2. Filaria sanguinis hominis.
3. Filaria medinensis.
4. Dochmius duodenalis.
5. Ascaris lumbricoides.
6. Ascaris mystax.
7. Eustrongylus gigas.
8. Trichocephalus dispar.
9. Oxyuris vermicularis.

I.—TRICHINA SPIRALIS.

SYNONYMS—Flesh worm, Pseudalius trichina.

LARVA—Muscle trichinæ, Encysted trichinæ, Flesh worms.

I. GENERAL CHARACTERS—

Trichina spiralis is a very minute worm, the male and female being distinct.

1. SIZE.—Male, $\frac{1}{18}$ inch; female, $\frac{1}{8}$ inch long.

2. STRUCTURE—

The *Head.*—Narrow, pointed, unarmed, with a simple central oval aperture.

The *Body* is thread-like, bent upon itself, thicker behind than in front, and in both male and female the hinder part of the body is straight. In the male, however, it has a short, bilobed caudal appendage, between the lobes of which is the anus. The testes are convoluted tubes. The female is about $\frac{1}{8}$ of an inch long, rounder and shorter behind than the male. There is an ovary, vagina, uterus, and the genital orifice is near the head.

The *Ova* are $\frac{1}{170}$ inch long, and are hatched within the parent (ovaviviparous).

LARVAL FORM—the trichina of muscle—is a very small worm, about $\frac{1}{30}$ inch long, coiled up in a spiral manner within a fibrous capsule or cyst, the long axis of which lies in the long axis of the muscular bundles. A single capsule may contain two or more larvæ and there may be as many as 325,000 of these capsules in an ounce of meat. They are especially common in the abdominal and thoracic muscles and appear as whitish spots from the cyst being often calcified towards the poles. This small worm has a digestive system and an imperfect sexual apparatus.

II. LIFE HISTORY—

When a piece of meat affected with trichinæ is eaten by an animal, the capsules are dissolved, and the embryo parasites which they contain are liberated. These mature in a day or two in the intestinal canal of the host. The sexes unite and give birth to ova and embryoes; a single ova producing over 1000 embryoes, and a single female discharging over 16,000 ova.

The embryoes migrate from the intestine to the striped muscles, passing through the intestinal walls, but it is not clear how they reach the muscles — possibly through the peritoneal cavity or through the blood and lymph.

Once in the muscles the embryoes penetrate the primitive bundles, reduce their contents to debris and soon become mature muscular trichinæ, forming cysts, part of which is made up of a chitinous secretion of the parasite, part by a wall of fibrous tissue formed of the perimysium of the muscle bundles. These cysts, as above stated, may become partly calcareous, giving rise to white shining spots in the muscle. They may remain quiescent for years.

INTERMEDIATE HOST.—The trichinæ are found in pigs, rabbits, sheep, dogs, rats, mice. The pig gets them from the rat, which acquires them from human fæces.

HOST. — Man, finding their way into his body through eating uncooked pork.

HABITAT.—The adult worm only inhabits the intestine and only lives for a few weeks.

III. EFFECTS—Trichinosis.

There is no marked effect unless the embryoes migrate in large numbers, when they cause fever, the muscles swell, become œdematous, and partial paralysis of the limbs is the result.

II.—FILARIA SANGUINIS HOMINIS.

SYNONYMS—Filaria Bancrofti, Filaria cystica, Trichina cystica.

I. GENERAL CHARACTERS—

The mature forms are rarely seen. The sexes are distinct. The male is said to be smaller than the female and lives in the same vessels.

"The female is described as a small slender hair-like worm with a club-shaped head, a narrow alimentary canal, a two-horned uterus usually full of embryoes. These are discharged through the vagina, which opens near the mouth."

The embryoes measure about $\frac{1}{70}$ inch long, have a rounded head, a tapering tail, and are enclosed in a fine membrane which does not burst, but which elongates as the embryo uncoils itself, thus forming a delicate sheath to the embryo.

II. LIFE HISTORY—

The adult worms inhabit the lymphatics. The embryoes gain access to the stomach of their host through drinking water; they thence make their way to the vessels. Here the sexes unite, and the embryoes are discharged into the lymphatics. In the day-time the embryoes are found in the lymphatics, but at night they crowd the blood stream. They cannot, however, undergo further development in man, and hence are taken up by a species of mosquito, in the body of which the embryoes are matured. When the insect dies, they find their way to water, thence to the stomach of their host, and, on reaching the blood and lymphatic system, form the mature filaria.

INTERMEDIATE HOST.—Mosquito.

HOST.—Man.

HABITAT.—The embryo, called filaria sanguinis hominis —in the blood and urine; the mature form, called filaria Bancrofti—in the lymphatics.

III. EFFECTS—

Chyluria — chyle in the urine. Lymph-scrotum and elephantiasis, from plugging of the lymph channels.

III.—FILARIA MEDINENSIS.

SYNONYMS—Dracunculus medinensis, Dracunculus Persarum, Guinea worm.

I. GENERAL CHARACTERS—

The sexes are distinct, male and female. The female alone has as yet been fully described. It is a fine thread-like worm, 1 to 6 feet long.

The *Head* is rounded, with a single central mouth, and four papillæ.

The *Body*, cylindrical in shape, has a firm chitinous cuticle, and a curved tapering tail. The intestinal canal is single—there is no anus—the uterus fills nearly the whole body.

The male is much smaller than the female. Embryoes have no envelope, and are born alive.

II. LIFE HISTORY—

The embryoes are found in water; they infect the bodies of certain crustacea. They thus gain entrance into the human intestine, and make their way to the skin of the legs and feet; and there causing ulceration, are discharged, and thus get access to the intermediate host.

INTERMEDIATE HOST.—Certain crustacea.

HOST.—Man.

HABITAT.—Soles of the feet, legs.

III. EFFECTS—

Ulceration of feet and legs, from escape of ova.

The Guinea-worm disease of India, etc., is the same disease as the Dracontiasis of Plutarch. The endemic affection described by Moses as due to the fiery serpents was probably owing to this parasite.

IV.—DOCHMIUS DUODENALIS.

SYNONYMS—Ankylostoma duodenale, Strongylus duodenalis, Sclerostoma duodenalis.

I. GENERAL CHARACTERS—

This is a small worm which infects the upper part of the small intestine, and sucks blood like a leech, and hence causes great anæmia—known as Egyptian chlorosis.

SIZE.—The male is about ⅜ inch long; the female, somewhat larger.

STRUCTURE—

The *Head* is pointed and bent backwards. The mouth is cleft-like, and has two chitinous lamellæ. The ventral lip has four curved conical teeth; the dorsal lip has two straight teeth.

The *Body* of the male ends behind in a trilobed, dilated extremity, with two thin spicula. The female ends behind in a sharp-pointed, awl-shaped spine. They are viviparous.

Ova.—Oval in shape.

II. LIFE HISTORY—

The embryoes pass through their first stage of development in the intestinal canal of man. They thence pass into dirty water, and back to their host.

III. EFFECTS — They cause great irritation, ulceration, hæmorrhage, anæmia.

IV. DISTRIBUTION—Italy, Egypt, South America.

V.—ASCARIS LUMBRICOIDES.

SYNONYMS—Round worm, Lumbricus teres hominis.

I. GENERAL CHARACTERS—

These parasites closely resemble the common earth worm.

SIZE. — The male measures about 4 to 6 inches; the female, 10 to 12 inches long.

STRUCTURE.—This parasite is a broad, smooth, fusiform, translucent, brown or red coloured worm, with fine circular striæ. Its anterior extremity has a three-lobed mouth. The tail is bluntly curved in the male, and has a double spicula near its end.

Ova, oval shaped, $\frac{1}{300}$ to $\frac{1}{150}$ inch in diameter, and have a hard shell and an albuminous envelope.

II. LIFE HISTORY—Not fully known.

HOST.—Man, pig.

INTERMEDIATE HOST.—Not required.

HABITAT. — The ileum, colon, also mouth and nose. They are passed by the fæces, or are vomited.

III. EFFECTS—

Little, except in children. In rare cases get into the bile duct. They may cause perforation of the intestinal wall, and ulceration.

VI.—ASCARIS MYSTAX.

SYNONYMS—Ascaris alata, Ascaris cati, Ascaris teres felis.

I. GENERAL CHARACTERS & LIFE HISTORY—

Smaller than the common round worm.

The ova contain the living embryoes, which, after being hatched outside the body, reach their host—cat, man—and there produce sexually mature worms.

VII.—EUSTRONGYLUS GIGAS.

I. GENERAL CHARACTERS—

They are reddish-brown coloured worms, with a cylindrical body, thicker at the posterior than at the anterior end, or head, which is obtuse and has six papillæ. The male is about 1 foot long; the female, 3 feet or so.

INTERMEDIATE HOST.—Some fish.

HOST.—Carnivora.

HABITAT.—Pelvis of the kidney.

VIII.—TRICHOCEPHALUS DISPAR.

SYNONYMS—Trichocephalus hominis, Trichuris, Ascaris trichiuria, Whip worm.

I. GENERAL CHARACTERS—

The whip worm is a very common intestinal parasite, characterised by the long, thin, thread-like head, attached to a thicker part—the body. The head burrows into the intestinal wall. The male measures about 1½ inches long; the female, about 2 inches.

Ova, about $\frac{1}{500}$ inch, and have a thick brown shell, knobbed at each end. They are very resistant.

II. LIFE HISTORY—

The ova are partly developed in water, in warm weather. They find their way to the intestine and there develop into mature whip worms. They pass the whole of their mature existence in human intestine.

INTERMEDIATE HOST.—Not required.

HOST.—Man.

HABITAT.—The cæcum and upper part of the colon.

III. EFFECTS—Harmless.

IX.—OXYURIS VERMICULARIS.

SYNONYMS—Thread worm, Ascaris vermicularis.

I. GENERAL CHARACTERS—

This is a small worm, with white, silvery, and shining cuticle. The head is fusiform, and has lobed appendages on each side, and three papillæ around the mouth.

The male measures about $\frac{1}{8}$ inch, and has an obtusely pointed tail; the female, $\frac{3}{8}$ inch, and the tail is long and slender.

Ova, $\frac{1}{900}$ inch—have dense shell.

INTERMEDIATE HOST.—Not required.

HOST.—Man; but especially seen in poor under-fed children.

HABITAT.—Cæcum, and pass to rectum, also to vagina. They pass the whole of mature existence in human body, and are acquired by drinking water. The ova are often found under the finger nails, and thus again gain entrance into the mouth.

II. EFFECTS—

Great irritation about anus, itching of nose, often convulsions and other nervous symptoms in children.

II. Epizoa — External Parasites — belong to the Class Arthropoda.

I.—ARACHINDA.

These parasites infect both man and other animals.

1.—ACARUS SCABIEI.

Synonyms—Sarcoptes scabiei, Sarcoptes hominis.

I. General Characters—

The body is oval in shape with a distinct head and four pairs of anterior, and four pairs of posterior, limbs. The male is smaller than the female, and the four anterior limbs each have a sucker, and the hinder pair of the posterior limbs also have suckers. In the female the four anterior pairs have suckers, the four posterior pairs have pointed bristle-like ends.

II. Life History—

The male lives upon the skin and is rarely seen. The female burrows beneath the skin, making a small tunnel in which she deposits her eggs. These channels are seen as black lines under the epidermis, and the female herself is generally found lodged at the far end of the tunnel. The epidermis falls off and the ova develop, reach the surface, and liberate the young.

III. Effects—

Inflammation, great itching, leading to scratching and eruptions.

II.—ACARUS FOLLICULORUM.

SYNONYMS—Demodex folliculorum, Face mite.

This is a small parasite with four pairs of feet. It is found on the face, and appears as little black specks. It inhabits the sebaceous follicles near the nose and external auditory meatus.

About 10 per cent. of French people are said to be affected by this parasite.

III.—PENTASTOMA DENTICULATUM.

SYNONYM—Pentastoma tænioides.

The LARVAL forms usually occur encapsuled in the liver, and are known as pentastoma denticulatum. They are about $\frac{1}{5}$ of an inch long and have many segments, in each of which are stomata, four hooklets with chitinous sheaths. In man they are usually dead and calcified.

The ADULT, known as pentastoma tænioides, inhabits the nasal cavity of the dog, and is similar to the larval form, but larger—the male being 1 inch, the female 3 inches, long. They have no hooks round the mouth.

The ova pass from the dog's nose on to grass, etc., and thus gain access to the stomach of hares and rabbits.

IV.—LEPTUS AUTUMNALIS.

Harvest bug.

The harvest bug is a small reddish parasite which invades the legs, burrows its way into the skin, causing great itching. It infects man, dogs, cats, rabbits, etc.

II.—INSECTA.

These are an unimportant group, and require but a passing notice.

I.—PEDICULI—LICE.

1. Pediculus Capitis (Head louse).—Lives amongst the hair. The ova form the *nits*—white spots seen on the hair—and are covered by a chitinous sheath. The young are like the adult forms.

2. Pediculus Vestimentorum.—Is larger than the pediculus capitis. The ova are deposited in the clothing.

3. Pediculus Pubis (Crablouse).—Is smaller than the last form and has long curved claws, with which it clings to the hairs. It occurs where there are short hairs as in the pubis, in the axilla, etc.

II.—PULEX IRRITANS.

The Common Flea.

The larvæ occur in the dust of the floor, on dry ground, etc.

III.—PULEX PENETRANS.

Sand flea, Jigger, Chigœ.

The female is very similar to the common flea; but it penetrates the skin of the soles of the feet, swells up into a sac, and causes very painful inflammation.

IV.—CIMEX LECTULARIUS.

Acanthia lectularia—Common bug.

Chiefly found in beds and bedding, and makes its way to the skin to suck blood, upon which it lives.

II.—VEGETABLE PARASITES.

I. DEFINITION—

VEGETABLE PARASITES belong to the lowest classes of the Vegetable Kingdom, and are known as Bacteria, Micro-organisms or Microbes, etc.

II. GENERAL CHARACTERS—

The vegetable parasites are Thallophytes—having no distinction between stem and leaves. They have, moreover, no chlorophyll, and hence are fungi, not algæ.

III. DIVISION—

There are three great classes—

1. SCHIZOMEYCETÆ—Bacteria.
2. BLASTOMYCETÆ—Yeasts.
3. HYPHOMYCETÆ—Moulds.

The Schizomycetæ are far the most important group as regards diseases, some of the most formidable scourges being associated with them.

The Blastomycetæ and Hyphomycetæ, on the other hand, exert chiefly a local action.

I.—SCHIZOMYCETÆ.

(Bacteria.)

($\sigma\chi\iota\sigma\tau o\varsigma$—Fission fungi).

I. GENERAL CHARACTERS—

1. SIZE.—The fission fungi or bacteria are exceedingly small microscopic organisms, being usually about $\frac{1}{10}$ inch in diameter—the largest forms reaching a diameter of two inches.

2. SHAPE.—Bacteria are rounded, oval, thread-like or rod-like in shape, and may have cilia, usually terminal in position—these, however, are often wanting.

3. STRUCTURE.—The schizomycetæ are non-nucleated uni-cellular plants, usually colourless, having no chlorophyll, but composed of a homogeneous or granular protoplasm, with a distinct cell wall.

(1) The *Protoplasm* differs from ordinary animal and vegetable protoplasm in its staining reactions, and is known as myco-protein.

(2) The *Cell-wall* consists of a substance closely allied to cellulose.

(3) The *Granules* within the cells are of the nature of fat, starch, etc.

(4) The *Inter-cellular Substance*, when present, may be formed by the softening of the outer part of the cell. It is frequently brightly pigmented, red, blue, or black in colour, though the cells themselves are colourless.

II. REPRODUCTION—

1. By **Fission.**—In this, the usual mode of multiplication of bacteria, the cells elongate and are subdivided by a transverse septum into two—the cells thus formed either separating or remaining united to form a chain.

In the rounded forms the fission may take place in several directions.

Some forms remain united by a gelatinous envelope, forming masses called *Zoogloea masses.*

2. By **Spores.**—This is the second mode of reproduction and occurs by—

(1) ENDOSPORES.—In this case part of the protoplasm of the cell becomes condensed into minute, clear,

rounded or oval globules—the endospores—a single spore being thus formed in each cell. These spores may either be discharged by the rupture of the cells, or they may lie dormant within the cells for long periods of time.

(2) Athrospores. — In this case the spores are not produced within the cells, but independent cells enlarge, the rest undergoing no change.

Spores are far more resistant to injurious agencies than are the cells themselves.

III. Classification—

Several modes of classification have been suggested but the classification according to form is the most convenient.

1. Cocci.—Spherical forms—
   - (1) Diplococcus—in pairs.
   - (2) Tetracoccus—in fours.
   - (3) Streptococcus—in chains.
   - (4) Staphyloccus—in clumps, like bunches of grapes.
   - (5) Sarcina—in packets.

2. Bacilli.—Rod-like forms—
   - (1) Bacillus—straight rods.
   - (2) Leptothrix—thread-like, without a sheath.
   - (3) Cladothrix—thread-like, with a sheath.

3. Spiro-bacteria.—Spherical forms—
   - (1) Spirochæta—flexible, with wide thread.
   - (2) Spirillum—stiff narrow threads.
   - (3) Vibrio—corkscrew-like.

IV. DISTRIBUTION—Bacteria are found—

1. In the air, water, on the surface of the earth.

2. On the exterior of bodies of animals, in the alimentary and respiratory tracts—not in the urinary tract.

3. Probably do not exist in healthy tissues and juices. (See page 192.)

V. CONDITIONS OF LIFE AND GROWTH—

1. Foods—

1. WATER.—All forms require moisture.

2. ORGANIC MATTER.—Since these micro-organisms have no chlorophyll, they cannot obtain carbon from carbonic acid, nor nitrogen from the more simple substances; they therefore require complex compounds, such as proteids, carbo-hydrates, etc. Some forms can split up ammonia; others can grow in fluids of very simple composition—Cohn's fluid. (See page 190.)

3. OXYGEN—

(1) Some forms require oxygen—*aërobic.*

(2) Others can live without oxygen—*anaërobic.*

4. ALKALINE AND NEUTRAL MEDIA are most favourable to the growth of bacteria. Acid media favour growth of moulds and yeasts.

2. Temperature.—The range of temperature favourable to life and growth of bacteria is very limited. Each form grows best at some one temperature; thus, bacteria associated with disease flourish most at the temperature of the body.

(1) Effects of *low* temperatures.—At freezing point, or 5° C., the growth of bacteria is arrested, but the

germs are not destroyed. Some forms may be frozen to ·110 without killing them.

(2) Effects of *high* temperatures. — 60° C. kills most bacteria, though spores are more resistant, requiring a temperature of 120° to 130° to destroy them.

Dry heat at 100° C. kills the spores of some bacilli in one and a half hours.

Dry heat at 100° to 115° C. kills some moulds in one and a half hours.

Dry heat at 140° C. kills bacillus anthracis in three hours.

Steam at 100° C. kills most forms in half-an-hour.

Moist heat at 57° C. destroys all forms.

3. Other Conditions—

(1) Strong sunlight arrests growth.

(2) Active movements check, while rest is favourable to growth.

(3) Electricity stops growth.

(4) Chemical agents—

(*a*) *Antiseptics* are those agencies which hinder the growth but do not destroy micro-organisms and their spores.

(*b*) *Disinfectants* are those substances which kill micro-organisms.

Examples—

1. Corrosive sublimate—

1 in 1,000,000 hinders growth.
1 in 300,000 arrests growth.
1 in 20,000 destroys growth.

2. Oil of mustard—
   1 in 330,000 hinders growth.
   1 in 33,000 arrests growth.
3. Carbolic acid—
   1 in 1250 hinders growth of bacillus anthracis.
   1 in 400 arrests growth.
   1 in 100 kills spores.

VI. Vital Activities—

1. Some forms of bacteria are the cause of special fermentation—zymogenic—*e.g.*, bacterium lactis, etc.
2. Some produce unorganised ferments—enzymes.
3. Some give rise to putrefaction, living on dead products—saprogenic—causing septic poisoning.
4. Some cause, or are closely associated with disease—pathogenic, causing septic infection.
5. Some produce substances closely allied to alkaloids called ptomaïnes (πτῶμα—a corpse)—sepsin, neurine, leucomaines.

VII. Method of Study—

In the study of bacteria and their life history we use the following methods—

1. Sterilisation.
2. Cultivation and Isolation.

1. Sterilisation.—Before cultivating growths of microorganisms we require to sterilise all apparatus, etc. This is effected—

1. By preventing the entrance of foreign germs by plugging tubes, etc., with cotton wool.
2. By heat—300° C. ensuring complete sterilisation.
3. By desiccation.
4. By chemical agents.

2. Cultivation—

I. Various Nutrient Media.

1. *Fluid Media*, such as—

   (*a*) Milk, urine, aqueous humor.

   (*b*) Meat infusions.

   (*c*) Cohn's fluid—

   Potassium phosphate, ·5.
   Magnesium sulphate, 1.
   Calcium phosphate, ·05.
   Ammonium tartrate, 1.
   Water, 100.

2. *Solid Media—e.g.*,

   (*a*) Peptonised meat broth added to gelatine to a suitable consistence — rendered alkaline or neutral, sterilised by heat and placed at a temperature of 20° to 25° C.

   (*b*) Agar-agar—a vegetable jelly procured from species of seaweed and used to thicken fluids.

   (*c*) Fresh cut surfaces of boiled potatoes.

   (*d*) Solidified and sterilised blood serum.

II. Method of Cultivation and Isolation.

To obtain a *pure cultivation* of an organism the *culture ground*, or medium, is inoculated by means of a wire with organisms, and from the growths thus obtained fresh inoculations on similar media are made. By this means the individual organisms are separated from all other forms and their mode of growth recognised. This is effected by—

(1) *Plate Cultivations.* — Some peptonised gelatine is gently heated till it melts, and is then, with due precautions, spread out in a thin layer upon a glass plate. It is now

inoculated at several points with the organisms to be studied and placed in suitable conditions to favour growth and prevent contamination. Other centres of inoculation are after a time, procured from these, and the organisms more and more isolated.

(2) *Test Tubes.* — These are partly filled with prepared jelly, inoculated with the organisms, plugged with wool, sterilised, placed in an incubator, at suitable temperatures, and their growth studied.

(3) *Cover Glass.*—A thin film of blood is spread upon a glass slide, dried, sterilised, and inoculated with the required growth.

(4) *In Animals.*—An animal is inoculated with bacteria. These increase within its body, and fresh inoculations are then made from it to other animals, thus passing the virus through several growths, till a pure cultivation is the result.

III. Mode of Distinguishing the various Forms of Bacteria when under Cultivation.

1. By their *mode of growth* — some forms growing in heaps, some branching.

2. By the *temperature* and *time* required for their growth.

3. By the changes which they produce in the *nutrient media*—

(1) Some forms, for example, liquify jelly.

(2) Some forms produce colours, odours, gases, and fluoresence.

VIII. Relations to Human Body—

1. Distribution—

(1) As before stated, bacteria are found in all parts of the body exposed to air. They also occur in the respiratory and alimentary tracts, but not in the healthy urinary tract.

(2) Neither bacteria nor their spores, however, exist in the normal healthy tissues or fluids of the body, and such as do gain entrance are soon destroyed by the vitality of the tissues.

Proofs—

(*a*) If small portions of the living tissues be removed from the body, with proper care, and examined microscopically, they are found to be free from micro-organisms.

(*b*) Again, if the tissues thus removed be cultivated, no growth of organisms occurs.

(*c*) Many forms of bacteria can be injected into the blood and produce no effects; they cannot live in the tissues.

2. Mode of Entrance of Bacteria—

(1) By wounds and abraded surfaces.

(2) By the mucous membranes of the respiratory and alimentary tracts.

(3) Indirectly, in some mode not yet known.

3. Mode of Spread—

(1) Locally, through the local continuity of the tissues and lymphatic spaces—*e.g.*, erysipelas.

(2) By lymphatic vessels and glands—*e.g.*, anthrax.

(3) By blood-stream—*e.g.*, tuberculosis.

4. Mode in which the Tissues are kept free from Micro-organisms—

(1) Many of the micro-organisms that enter through the respiratory tract, are stopped in the inter-pulmonary bronchi.

(2) Those that gain entrance to the tissues are destroyed by the agency of leucocytes, or of connective tissue cells or cells derived from them, which, from the power they possess, are called *phagocytes* (φαγειν to devour).

IX. Relations of Bacteria to Disease—

In their relation to disease, micro-organisms may be divided into two groups :—

1. **Pathogenic.**—Those micro-organisms which, when inoculated into the system can, under suitable conditions, multiply within the tissues and fluids of the body, and either by their direct action, or by the virus to which they give rise, produce specific local or general effects. They cause Septic Infection, the virus being capable of spreading from animal to animal.

2. **Non-Pathogenic**—(Saprophytic).—These micro-organisms cannot multiply within the body, but can live upon dead tissues, and can produce fermentations and putrefactions. They may give rise to substances of the nature of alkaloids called ptomaïnes, which, when absorbed into the blood, set up septic poisoning—to be carefully distinguished from *septic infection*. Their effects vary according to the dose, being non-infective.

Evidence required to prove that a Disease is due to the Agency of a Micro-organism.

(1) The constant presence (in the disease in question) of definite micro-organisms in the tissues or juices of the animal affected.

(2) The suspected organism must be cultivated through several generations, and, when a pure cultivation has been thus obtained, must when inoculated produce the original disease.

(3) The virus must be capable of transmission from one animal to another, and must produce the disease.

(4) There should be an observed constant relation between certain lesions and certain forms of micro-organisms —thus, tubercle-bacillus is constantly found in tubercle nodules, etc.

X. Mode of Action of Bacteria in the Production of Disease—

This varies with different organisms, and in different diseases—

1. Some bacteria are said to produce their effects by removing oxygen, and thus preventing the due oxidation of the tissues; but against this is the fact that many pathogenic bacteria do not require oxygen for their growth.

2. Other forms act by causing obstruction to the blood or lymphatic channels.

3. Others, by direct local effects upon the tissues, causing irritation and inflammation.

4. Others, by the production of poisonous substances—pyogenic substances, alkaloids, ptomaïnes.

XI. Conditions Required for Production of Disease by Bacteria—

1. The virus must be of a certain amount, and must, in most cases, be capable of increase within the body.

2. There must be a suitable soil, or "*nidus*"—such as general lowered vitality, or local damage to tissues, etc.

Examples—

(1) If an animal have a bone fractured, healing will quickly occur, but if the animal be fed on putrid meat we get acute suppuration.

(2) Blistournage.—If the spermatic cord of an animal be crushed, it produces atrophy of the testicle; but if bacteria be injected into the blood, suppuration results.

3. The micro-organisms must be arrested in the blood or tissues. Active movements are unfavourable to their growth. Such arrest is, however, not required for all organisms.

XII. IMMUNITY FROM ACTION OF BACTERIA—

1. Natural Immunity (Spontaneous).—It is found that certain individuals, certain races, and certain animals are proof against certain forms of disease—*e.g.*,

(1) Small-pox affects negro races more than white races.

(2) Certain breeds of sheep are proof against anthrax.

(3) Micro-coccus tetragenus, when inoculated into white mice, causes pathogenic effects; when inoculated into field or house mice, it has no effect.

2. Acquired Immunity—

(1) A certain degree of immunity may be acquired by HEREDITY—*e.g.*, the introduction of measles amongst the South Sea Islanders at first caused great ravages, but the disease afterwards died out.

(2) A partial immunity can be acquired by improving the general health. Lowered vitality predisposes to disease.

(3) Immunity due to INOCULATION. When certain diseases are inoculated—*e.g.*, small-pox, or vaccinia—they protect the patient from further attacks, or at least modify future attacks.

Some diseases, so far from protecting from further attacks, render the person even more susceptible—*e.g.*, diphtheria, erysipelas, etc.

Some organisms seem to prepare the tissue for the action of other organisms—thus, whooping-cough is liable to be followed by measles.

3. Modification of Virus.—The activity of disease-producing microbes can be altered—

(1) By CULTIVATION in several *different media*—thus, bacillus anthracis grown in meat broth, loses much of its virulence if grown upon potatoes or in hay infusions.

(2) By changing the TEMPERATURE at which the organisms grow.

(3) By changing the supply of OXYGEN—thus, Pasteur cultivated the microbe of fowl cholera in chicken broth exposed to a free current of oxygen, and by this means attenuated the virus.

(4) Virulence may be modified by cultivating through *another animal*— thus, if rabbits are inoculated with the virus of hydrophobia and then killed, and the spinal cord removed, dried, and an emulsion made of it, an attenuated virus is obtained which, when inoculated, protects against the disease.

(5) Some organisms may have their virulence increased by certain modes of cultivation—thus, some microbes require to pass through the human body before reaching their full virulence.

XIII. ANTAGONISM OF BACTERIA —

Both inside and outside the body there is supposed to be a certain antagonism between different species of bacteria —thus, typhoid fever antagonises anthrax; a person suffering from small-pox is said not to be liable to scarlet fever; but as yet there is much doubt on these points, for several organisms will flourish side by side, provided they are furnished with proper nutriment.

XIV. CO-OPERATION OF BACTERIA IN THE PRODUCTION OF DISEASE—

A single organism may possibly constitute the specific virus of a disease, but to produce typical leisons the co-operation of several forms of microbes may be required,

either to prepare the system for the operation of the specific virus or to act along with it in the production of the disease.

Examples—

(1) Diphtheria is probably due to the combined action of a bacillus and a micro-coccus.

(2) Vaccinia is due to action of two organisms.

(3) In suppuration a number of different organisms are always present.

XV. Diseases Associated with the Action of Bacteria—

These diseases may be arranged in three groups—

1. Suppuration and Wound Infective Diseases.
2. Specific Infective Diseases—Acute Specific Fevers.
3. Chronic Infective Inflammations—Granulomata.

I.—SUPPURATION AND WOUND INFECTIVE DISEASES.

These are the result of septic and infective processes in wounds, and are due to the action of micro-organisms.

1. ACUTE SUPPURATION.

Acute suppuration occurs in relation to wounds and to abscesses, and owes its origin to the action of micro-cocci, which are always found in great abundance in pus of these wounds. Experiments, however, have shown that other agents, such as turpentine, cadaverin, etc., can produce suppuration.

Organisms in Pus.—Pus, which differs from ordinary inflammatory exudation in not being spontaneously coagulable, is rarely found in internal organs not exposed to air. Its presence in abscesses is indicative of the fact that microbes have gained access to the affected part.

The most common organisms found in pus are—

(1) Staphylococcus pyogenes aureus.
(2) Staphylococcus pyogenes albus.
(3) Streptococcus pyogenes.

1. Staphylococcus Pyogenes Aureus.

Characters.—This micro-coccus is spherical in form, about ·8 to ·9 m. in diameter, and usually occurs in masses, though it may exist in pairs or chains. It stains easily with logwood and carmine, and has great vitality, resisting for a long time the action of heat and drying.

Distribution.—It occurs in all inhabited places, and has been cultivated in air, water, etc. It is found in pus, in boils, in carbuncles, in suppurating glands, etc.

Cultivation.—Staphylococcus pyogenes aureus grows in nutrient jelly or agar-agar, and flourishes best at a temperature of 30° C. It can liquefy jelly, and in the presence of oxygen produces a dull yellow pigment. Little or no oxygen, however, is needed for its growth. It gives rise to a marked odour, especially when grown upon potatoes.

Effects.—Staphylococcus pyogenes aureus produces lactic and butyric acid fermentation in milk. It peptonises albumen, but does not produce ptomaïnes. When injected into man it causes suppuration and necrosis of the part; all animals, however, are not affected by it.

2. Staphylococcus Pyogenes Albus is similar to the last form, but does not produce colours when grown in nutrient media.

3. Streptococcus Pyogenes.

Characters. — In the tissues streptococcus pyogenes occurs as diplococci, or in small rows. In size it measures about ·6 to ·7 m.

Cultivation. — Streptococcus pyogenes grows upon nutrient jelly, in blood serum, in meat broth. It is of slow growth, does not liquefy jelly, and flourishes best at a temperature of 95° to 98° C.

Effects.—Streptococcus is the most common organism met with in pyæmia, occurring in spreading inflammations and suppurations.

It appears to be a similar, if not an identical, organism with that found in ulcerative endocarditis and erysipelas.

2. WOUND INFECTIVE DISEASES.

1. Definition. — These are general systemic blood-poisonings, associated with wounds, or with internal leisons which afford a suitable nidus for the lodgment of a specific virus, and may occur in three modes—

(1) The micro-organism present in the pus in septic wounds may produce poisonous substances—ptomaïnes, etc.—which when absorbed into the blood cause septic poisoning—Toxæmia.

(2) The organisms, which are the active agents in the production of suppuration, may themselves enter the circulation and set up foci of suppuration throughout the body.

(3) The original wound may serve as the point of entrance of pathogenic bacteria, which, finding a suitable nidus, give rise to these effects in the organs, etc., to which they gain access. It is difficult to decide between these three modes of blood-poisoning.

2. Division.—These wound infective diseases include—

1. Septicæmia and Pyæmia.
2. Spreading Traumatic Gangrene.
3. Wound Erysipelas.

I.—SEPTICÆMIA AND PYÆMIA.

There is no essential difference between these two forms of general blood-poisoning—*Septicæmia* being the term used when there are no secondary foci of suppuration, whereas *Pyæmia* indicates the existence of secondary abscesses in various parts of the body.

1. SEPTICÆMIA.

I. Definition—

Septicæmia (as above stated), is the name given to those cases of blood-poisoning in which there are no secondary abscesses formed.

II. Division—There are two forms of Septicæmia.

1. Septic Poisoning—Toxæmia—Septic Intoxication—Sapræmia—which is a non-infective disease due to absorption into the blood of the chemical products, poisonous alkaloids, ptomaïnes, leucomaines, etc., produced in the process of putrefaction. The effects vary with the dose of the poison absorbed, for the virus has no power of self-multiplication in the blood or tissues. Little is as yet known of the nature of ptomaïnes, etc.

2. Septic Infection—Septicæmia proper—differs from septic intoxication in being intensely infective. The virus, when once introduced into the blood, has great powers of multiplication, so that even a minute quantity of the poison may give rise to serious effects.

III. SYMPTOMS—

The clinical symptoms of both forms of blood-poisoning are practically the same—viz., acute fever, rigor, great prostration, dry tongue, rapid emaciation, rapid feeble pulse, high temperature 117° C., vomiting, diarrhœa, a jaundiced condition of the skin, petechiæ, albuminuria.

In the non-infective form death is characterised by dyspnœa, collapse, and cardiac failure; in the infective form there is a semi-comitose condition.

IV. MORBID ANATOMY—After death, there is feeble rigor, quick decomposition.

1. THE BLOOD.—Dark coloured, usually tarry, fluid, though often clotted.

2. INTERNAL ORGANS.—There is blood-staining of endocardium and of the lining of great vessels, petechiæ of pleura and pericardium, and hypostatic congestion of the lungs. The spleen is soft, swollen, pulpy. Liver and kidneys are swollen, congested, and in a state of cloudy swelling.

2. PYÆMIA.

I. DEFINITION—

Pyæmia is essentially the same disease as Septicæmia, but the poison absorbed into the blood not only causes a general disease, but also gives rise to the formation of secondary foci of suppuration—*Metastatic Abscesses*—in different parts of the body.

The nature of the poison causing pyæmia is not certainly known, but is probably of the nature of ptomaïnes.

II. CAUSES—

1. Pyæmia is associated with septic wounds, with gastric ulcers, etc.

2. In some cases there are no wounds—*e.g.*, in infective endocarditis and infective peritonitis. It may be secondary to suppuration in the kidney, middle ear, etc.

III. Symptoms—

The clinical symptoms of pyæmia are very similar to those of septicæmia, the irregular temperature being especially characteristic.

IV. Morbid Anatomy—

(*a*) In or near the Wound.

1. The *Wound* itself has an unhealthy, sloughly appearance, and contains an accumulation of decomposing pus.

2. The *Veins* leading from the wound are in a state of suppurative phlebitis, containing thrombi which are undergoing infective puriform softening.

(*b*) In Internal Organs—

1. There is hypostatic congestion of the lungs, enlarged and pulpy spleen, and the liver and kidneys are in a state of cloudy swelling.

2. *Metastatic Abscesses* are found in different situations, and are of two kinds—

(1) Localised abscesses.
(2) Diffuse suppurations.

Metastatic Abscesses—

1. *Characters*—

(1) Localised Metastatic Abscesses are mostly found near the surface of the affected organ. They are usually multiple in character, and vary in size from a pea to a walnut. They contain greenish-looking, offensive-smelling pus, and are surrounded by a zone of congestion.

(2) The Diffuse Form occurs as a diffuse suppuration of the subcutaneous tissues and intermuscular septa, and also in serous and synovial membranes.

2. *Sites.*—Metastatic abscesses may occur in any vascular organ, but the usual sites are—

(1) The lungs — most commonly, in the posterior part of the lower lobes. The intervening lung tissue may be in a state of pneumonic consolidation. The process, moreover, often spreads to the pleural sac. In various parts of the lung there are minute infarcts undergoing process of necrosis.

(2) Liver—especially when the portal vein is connected with the site of the leison.

(3) Heart—in the wall, especially near the base of left ventricle.

(4) Brain—usually one or two in number.

(5) Kidney—near the cortex.

(6) Spleen.

(7) Joints—in the synovial membranes.

3. *Causation—Mode of Formation.*—These secondary abscesses owe their origin either—

(1) To infective emboli, which have been carried from the primary leison by the blood stream, and, causing local infarcts, set up subsequent suppuration.

(2) In other cases there are no evidences of the formation of infarcts. The irritant in this case being carried to the affected spot by the blood, and finding a suitable nidus, settles down and produces secondary effects.

Capillary emboli, however, may possibly be the cause of the diffuse suppuration. The micro-cocci carried by the blood become lodged in the capillaries, grow in the endothelial cells, and give rise to local coagulation of blood, followed by decomposition and softening, thus setting up local suppuration of the vessel wall.

The diffuse suppuration is due to extension of small abscesses and to the confluence of several abscesses.

II.—SPREADING TRAUMATIC GANGRENE.

Spreading traumatic gangrene is probably due to streptococcus pyogenes, though it is more than probable that it owes its origin to the action of more than one form of micro-organism. Some forms of gangrene are probably derived from the lower animals.

III.—WOUND ERYSIPELAS.

Wound erysipelas is usually a superficial inflammation, though sometimes a deep-seated cellulitis occurs. There is an intense red colour of the skin, with a raised border. It spreads rapidly and may give rise to blebs or bullæ. The epithelium usually desquamates.

Micro-cocci are found in the affected part, especially at the spreading edge and in the superficial lymphatic channels along which the infection spreads.

Erysipelas has been inoculated in rabbits by the injection of fluid from one of the bullæ. A streptococcus very similar to, if not identical with, streptococcus pyogenes of suppuration is always present.

II.—SPECIFIC INFECTIVE DISEASES—ACUTE SPECIFIC FEVERS.

Many of the acute specific fevers have been more or less definitely proved to be associated with the action of a virus of the nature of a micro-organism. In other cases, however, such as small-pox, scarlet fever, a specific organism has not yet been isolated.

As examples of specific infective diseases associated with micro-organisms, we may cite the following:—

I.—ANTHRAX.

I. DEFINITION—

Anthrax—also called Charbon, Malignant Pustule, Wool-sorters' Disease, Splenic Fever, Splenic Apoplexy—is a disease which especially attacks cattle, horses, sheep, deer—less commonly swine, dogs, etc.; but it also occurs in man, and is due to the agency of a specific bacillus—bacillus anthracis.

II. DIVISION—Anthrax, as it occurs in man and animals, may be—

(1) Local.
(2) General.

In man the disease manifests itself chiefly in the local form; in cattle, etc., the general form is more common.

III. CHARACTERS—

(1) In Animals.
(2) In Man.

1. Anthrax in Animals—

1. There is but little local leison. The chief changes are in the *Spleen*, which becomes like a mass of blood clot—hence the name, splenic apoplexy.

2. Hæmorrhages occur in such organs as lungs, wall of heart, cortex of kidney, brain and its membranes.

3. There are areas of inflammatory exudation—cellulitis, effusions into serous cavities.

4. The blood has a dark colour, and is crowded with bacilli.

5. Lymphatic glands are also affected—more especially in animals, not so much in man.

Situations in which the Bacilli are found—

(1) In the blood—appearing as rod-like bodies; they are in enormous numbers. No spores are formed in the blood.
(2) In serous effusions—in hæmorrhages.
(3) In the pulmonary capillaries.
(4) In venules of affected part.
(5) In the kidneys—especially in the Malpighian bodies.
(6) In the liver, and portal system.
(7) In the spleen—but somewhat difficult to detect.
(8) In the skin.
(9) In the pia mater.
(10) In lymphatic glands.

2. Anthrax in Man. — Anthrax occurs in persons working amongst hides, hair of cattle, etc.; hence in tanners, wool-sorters. The poison, however, can be carried by flies.

1. Mode of Inoculation—

(1) By wounds.
(2) By the respiratory tract.
(3) By the alimentary tract.

2. Division.—There are two forms in man—

(1) *Local Form—Malignant Pustule*—due to inoculation; hence it occurs on exposed parts—on the face, neck, lips, hands, arms. It commences as a small pimple surrounded by a bluish inflammatory zone. The pimple may or may not burst, but in two or three days it enlarges and fresh vesicles form—bullæ follow, and we get local gangrene and necrosis. General blood-poisoning may ensue. The bacillus is found in the superficial and subcutaneous lymphatics of the affected area, and afterwards in the blood, etc.

(2) *General Form.*—Rare in man, but may affect—

(*a*) The *Respiratory Tract.*—Wool-sorters' disease. The primary leison is usually in the lower part of the trachea and larger bronchi, where there are patches of intense swelling of the mucous membrane, with hæmorrhages and ulcerations. Great swelling of mediastinal glands, which, from hæmorrhages, look like blood-clots.

(*b*) The *Gastro-intestinal form* gives rise to diffuse inflammation, with partial detachment of mucous membranes. Hæmorrhages also occur in this situation. In these cases there are few bacilli in the blood, but they are found in great numbers in the bronchi and in lymphatic glands.

IV. CAUSE—

Anthrax is due to bacillus anthracis, which gains entrance into the body by the food, by the respiratory tracts, by wounds.

Bacillus Anthracis—

1. CHARACTERS—

(1) *Shape.* — As it occurs in the blood of affected animals, bacillus anthracis consists of straight rods joined end to end, with blunt, slightly curved extremities. Motionless—thus differing from bacillus subtilis. There are no spores in the blood; these have been seen in the kidneys only.

(2) *Size.* — They vary considerably—from 5 to 10 or 20 m. in length—1·2 m. broad.

2. CULTIVATION.—Bacillus anthracis will grow on gelatine, agar-agar, potatoes, in hay infusions, in aqueous humour. It liquefies jelly, and sinks to the bottom of the vessel in whitish masses. It requires oxygen for its growth. When cultivated in potatoes it gives rise to dry, creamy, yellow-coloured masses. When grown at a temperature of 15° to 42° C., it forms long filaments full of spores.

3. Reproduction—

(1) By spores, which form in the filaments as highly refracting rounded spots.

(2) In the living body neither spores nor filaments are found, but the rods multiply by division.

4. Inoculation.—The attenuation of the virus of anthrax has been effected by cultivation through animals, and also by cultivation on nutrient media, at a temperature of 42° to 43° C., for some days. By these means the virulence of the virus is modified and produces a "vaccine" which, when inoculated, gives a certain degree of immunity from the disease.

II.—TYPHOID FEVER.

I. General Characters—

Typhoid fever is an acute specific fever in which there is general blood-poisoning, septic in type, with waxy degeneration of the muscles, ulceration of the larynx, bronchitis, broncho-pneumonia, œdema of the lungs, and cloudy swelling of many organs.

II. Morbid Anatomy—

The characteristic leison of typhoid fever is to be found in the lymphoid tissue, especially in the solitary and agminated glands (Peyer's patches) of the small intestine, which become infiltrated with small round cells, and are surrounded by a zone of hyperæmia. The new formed tissue now either breaks down and undergoes *resolution* or tends to the formation of *typhoid ulcers*, which either cicatrise or result in perforation of the intestinal wall.

The spleen, mesenteric glands, and red marrow, are also affected.

III. CAUSE—

Bacillus of typhoid. Bacilli have been found not only in Peyer's patches, but also in the spleen, kidney, heart, blood, lymphatic glands, and in the roseolar spots on the skin.

From the constant relation of these bacilli to the characteristic leison of typhoid, they have been supposed to be the cause of the disease; but they have not as yet been successfully inoculated.

Typhoid Bacillus—

1. CHARACTERS.—It is described as a short thick mobile bacillus, longer than broad, with rounded ends, spores often being seen in the rods.

2. CULTIVATION.—It has been cultivated in nutrient media, but stains with difficulty, and cannot be inoculated in animals.

III.—RELAPSING FEVER.

(Famine Fever.)

I. GENERAL CHARACTERS—

This disease is characterised by an acute attack of fever with great prostration, followed by apparent recovery, but in a week or ten days the fever returns.

II. CAUSE—

An actively moving bacillus—spirochæta—has been found in the blood during the height of the fever, disappearing with the remission of the symptoms, and reappearing in the acute stage. No spores have been observed, and the spirillum has not been found in the tissues.

The disease has been inoculated in monkeys, and has been transmitted from man to man. The virus has not been successfully cultivated.

IV.—ASIATIC CHOLERA.

I. General Characters—

Asiatic Cholera is an acute disease in which there is intense diarrhœa, profuse watery discharges known as rice-water stools.

II. Morbid Anatomy—

The leison occurs in the lower part of the small intestine, in which are found patches of hyperæmia, minute ecchymosis, especially round Peyer's patches and solitary glands. There is swelling of lymphatic glands and a superficial necrosis of the mucous membrane of the intestine.

III. Cause—

A bacillus, called the comma bacillus, has been found in the intestine, in the discharges and mucous membrane, and also in the water which had been drunk by cholera patients.

Comma Bacillus—

1. Characters.— It measures about ½ to ⅔ the size of the tubercle bacillus—thicker, however, and curved like a comma; hence the name. When cultivated it sometimes assumes the spiral form.

2. Cultivation.—It will grow on almost any medium—a temperature of 30° to 40° C. being the best.

The growth commences as whitish spots, then the jelly liquifies and the mass sinks in the jelly, giving rise to a very characteristic appearance of a narrow funnel. It grows rapidly and is reproduced by athrospores. Drying is said to kill the cells in a few hours, but this is denied by some.

The bacillus has been inoculated into guinea-pigs, and has caused a disease similar to cholera. The contagion exists

in the dejecta of the patient and is spread by infection from water, etc.

The parasite can multiply outside the body, and in cholera districts has been found especially in water contaminated with organic matter.

V.—DIPHTHERIA.

I. General Characters—

Diphtheria is a spreading inflammatory disease, characterised by the formation of a *false membrane* on the fauces, tonsils, soft palate, and extending thence into the air passages.

The False Membranes are of two forms—

(1) The *Croupous*—when the epithelium of the mucous membrane is alone involved.

(2) *Diphtheritic*—when the deeper layers of the mucosa are affected.

The *Structure* of the false membranes varies somewhat, but they may be said to consist of altered epithelial cells which have undergone coagulative necrosis, and of a network of filaments of fibrin. When the membrane is stripped off, an inflamed surface is left beneath and the false membrane is reformed.

II. Cause—

The virus of diphtheria is as yet undiscovered, but probably there are two organisms concerned in its causation—a micro-coccus and a bacillus.

(1) In the meshes of the filaments of fibrin, many leucocytes and micro-cocci have been observed. They are also found in lymphatic glands of the neck, causing them to become enlarged.

(2) In the deeper layers of the false membrane, and in various internal organs, a bacillus is constantly present. It is almost the same size as the tubercle bacillus, but double its thickness. It has been cultivated in blood serum and when injected into the larynx or conjunctiva of the rabbit gave rise to the characteristic diphtheritic leison. It is, however, often absent in cases of diphtheria. When the micro-coccus is injected, an inflammation closely allied to erysipelas is the result.

VI.—LOBAR PNEUMONIA.

(Acute, Croupous.)

I. GENERAL CHARACTERS—

An acute inflammation of the parenchyma of the lung, leading to consolidation of large areas of lung tissue. It usually occurs on the right side, and chiefly affects the lower lobe.

There are three stages in the disease—

(1) The stage of engorgement.
(2) The stage of grey hepatisation.
(3) The stage of red hepatisation.

By many, lobar pneumonia is regarded as an infective inflammation of the lung, similar to erysipelas in the skin; by others it is said to be an acute specific fever, of which the inflammation of the lung is the local leison.

II. CAUSE—

Many micro-cocci have been found both in the lung tissue and in the sputum in cases of pneumonia, but two forms alone require note—

(1) The coccus of Friedländer.
(2) The diplococcus of Fränkel.

These are two distinct organisms, the latter being the more probable cause of the disease. When cultivated, it occurs as oval cells, usually in pairs, though they may be in chains, and are surrounded by capsules. In the tissues they often occur as lancet-shaped bodies. When cultivated at a temperature of 42° C., their virulence is soon destroyed. They retain aniline dyes and cannot be cultivated in gelatine, for they require a temperature that would liquefy jelly. When injected under the skin they do not cause pneumonia, but an attenuated virus injected into the lung usually does produce pleurisy or pneumonia.

The coccus of Friedländer, on the other hand, can be cultivated in gelatine and does not stain with aniline dyes. It is an oval or rod-shaped coccus, surrounded by a capsule, two or more cocci being contained in each capsule.

VII.—MALARIA.

I. General Characters—

Malaria — Ague — is an endemic fever occurring in marshy districts. It presents several varieties and is characterised by the regularity of the occurrence of the attacks, being quotidian, tertian, quartan, etc.

II. Morbid Anatomy—

1. Pigmentation of Organs—

The chief change in ague is the deposition within the affected tissues and organs of a dark brown or black pigment. This is especially seen—

(1) In the spleen, liver, lungs.
(2) In lymphatic glands.
(3) In the brain and spinal cord.
(4) In the skin and serous membranes, etc.

The pigment, which is derived from the hæmoglobin of the disintegrated red corpuscles, is deposited in the walls

of the arteries, and is carried to other parts by the leucocytes, giving rise to the pigmentation above noted.

2. Melanæmia—

This term is applied to the pigmented state of the blood found during life in patients suffering from ague. The pigment granules are seen both in the red and white corpuscles and in the blood plasma.

3. Other Changes—

(1) There is great destruction of the red corpuscles.
(2) A great enlargement of the spleen, and also to some extent of the liver.

III. Cause—

Plasmodium Malariæ — Hæmatomas Malariæ. — The exact cause of malaria is not known. It was at one time supposed to be due to a bacillus. It is now said to be caused by the plasmodium malariæ — hyaline, amœboid masses, with granular pigment, found in the red corpuscles of the blood. Other forms are also found, both in the corpuscles and free in the blood. The parasite causes great destruction of the red corpuscles and consumes the pigment.

III.—INFECTIVE GRANULOMATA.

I. Definition—

Granulomata — Infective Granulomata — are new growths, chiefly composed of tissue resembling granulation tissue; hence their name. They are closely allied to sarcomata, being made up of cells of various sizes embedded in a scanty matrix. They are Chronic Infective Inflammations, due to the action of a specific virus, probably of the nature of a micro-organism.

II. DIVISION—This group of new growths includes—

1. Tubercle.
2. Lupus.
3. Leprosy.
4. Glanders and Farcy.
5. Syphilis.
6. Actinomycosis.

III. GENERAL CHARACTERS — The granulomata are—

1. SPECIFIC INFLAMMATORY PROCESSES, showing special characters—

(1) In the *Course* they pursue — being progressive, persistent growths; and

(2) In the *Structures* produced — these being midway between granulation tissue and tumours, but dominated in each case by the specific virus to which they probably owe their origin.

2. They are INFECTIVE—and many of them inoculable —the contagion being a vegetable micro-organism which produces well marked specific local leisons.

They tend to spread through the ordinary channels—the blood-vessels and lymphatics, and have long periods of evolution—the virus persisting *per se*, or in its effects, for an indefinite period of time.

I. — TUBERCLE.

(Tuberculosis.)

I. DEFINITION—

TUBERCLE or TUBERCULOSIS is the name given to an infective inflammatory disease, characterised by the formation of nodules — tubercle nodules in certain tissues and organs—due to the agency of a specific virus—the tubercle bacillus.

II. DIVISION—There are two forms of tuberculosis—

1. LOCAL TUBERCULOSIS — where the leison is at first limited to one or more small areas.

2. GENERAL TUBERCULOSIS — where the leisons are multiple and general in distribution. It may be subdivided into—

(1) *Acute General Tuberculosis* — in which case there is a simultaneous and widespread formation of tubercles in many organs.

(2) *Chronic General Tuberculosis*—in which the disease is characterised by the formation of few leisons, which run a chronic course.

III. GENERAL CHARACTERS—

Tuberculosis is one of the most widespread of diseases, affecting alike man and animals — being one of the most fatal of human diseases. It commences as a local leison, characterised anatomically by the formation of masses called *Tubercles*, due to the local action of the tubercle bacillus — the results of the activity of which are called *Tubercular*, and are two-fold—

1. TUBERCULAR NODULES OR GRANULATIONS.
2. TUBERCULAR INFILTRATIONS.

IV. STRUCTURE — The anatomical products of tuberculosis are—

1. Tubercular Nodules, which are of two kinds—

(1) Grey tubercles—grey granulations—miliary tubercles.

(2) Yellow tubercles.

1. GREY TUBERCLES—GREY GRANULATIONS—

MACROSCOPIC STRUCTURE.—Grey tubercles—grey granulations — are semi-translucent, rounded, greyish-looking,

non-vascular nodules, exceedingly minute—$\frac{1}{16}$ to $\frac{1}{8}$ inch in diameter—though they are frequently confluent. Sometimes they are larger, firmer, shot-like, and circumscribed.

Microscopic Structure.—These tubercles are seen to consist of a number of confluent masses, composed of *tubercle follicles* or *elementary tubercles*. They are non-vascular, irregular in shape, and are surrounded by a zone of leucocyte infiltrations. There are three forms—

(1) *Typical Giant-celled Tubercle Follicles.*—Composed of three distinct elements—

(*a*) *Giant Cells* in the centre of the nodule.

(*b*) *Epithelioid Cells* around these—forming a middle zone.

(*c*) *Leucocytes* in a lymphoid stroma, forming the peripheral zone.

(2) Tubercles composed of masses of cells closely resembling granulation tissue. The cells, however, have a relatively large amount of protoplasm round the nucleus, and are called *Epithelioid Cells* from their resemblance to epithelial cells. This form is especially met with in the pia mater and in serous membranes.

(3) Localised masses of inflammatory products, consisting partly of leucocytes and partly of proliferated tissue of the organ in which the tubercle occurs. There are, however, no epithelioid and no giant cells. They tend to caseate early.

2. Yellow Tubercles—

These are merely a transition form of grey granulations. They are larger in size, softer, yellow in colour, and are due to commencing fatty degeneration of the grey tubercles. There are all gradations between the two forms.

2. Tubercular Infiltrations—

In this case there are diffuse inflammations—not nodular in character—composed of non-vascular masses of cells, separated from each other by round-celled infiltrations, as seen in tubercular peritonitis and meningitis. They are diffuse inflammations—acute or chronic—with the formation of granulation tissue, but characterised by their tendency to caseation. There are, however, usually some tubercular follicles scattered through the granulation tissue. These tubercular masses, therefore, can be distinguished from ordinary granulation tissue by—

1. The nodular arrangement of their elements.
2. The presence of more giant cells.
3. Their tendency to caseation and necrosis.
4. The presence of the tubercle bacillus.

V. Development of Tubercle Follicles—

When the tubercular bacillus gains access to a tissue, it, under favourable circumstances, sets up changes in the cells of the part—they swell up, their nuclei proliferate, and give rise to giant cells and epithelioid cells. This proliferation of cells is accompanied by inflammation, increased vascularity, and leucocyte infiltration.

As above stated, the typical tubercle follicle consists of—

(1) Giant cells.
(2) Epithelioid cells.
(3) Lymphoid tissue—granulation tissue.

1. Structure of—

(1) *Giant Cells.*—Fully formed giant cells are large, branching, multi-nucleated masses of protoplasm, highly granular, often vacuolated—the nuclei being arranged at the margin of the cell. These giant cells, however, are not characteristic of tubercle alone; they are found under

many other conditions—*e.g.*, in myeloid sarcoma, granulation tissue, etc.

(2) *Epithelioid Cells.*—These are smaller than the giant cells; they have one or more nuclei, are like epithelial cells, and form a zone round the giant cells.

(3) *Lymphoid Tissue.*—This is found at the margin of the tubercle nodule, and consists of round cells—leucocytes—in a homogeneous network, forming a mass of granulated tissue ill-defined from the surrounding tissue.

2. Source of these Various Constituents of the Tubercle Follicle.—This is as yet disputed—

(1) Some holding that these cells arise from connective tissue cells.

(2) Others, that they come from leucocytes.

(3) Others, that they arise from epithelial cells.

It is more than probable that they are derived from all these three sources.

Experimental Evidence—

Baumgarten and others have produced structures similar to tubercle by irritating the cornea of rabbits by hairs, etc., but the products are local and not infective. If tubercle bacillus be injected into the same situations typical tubercle follicles are formed, but the infection spreads to neighbouring glands and causes general tuberculosis.

VI. Sites of Tuberculosis—It occurs in—

1. Skin and subcutaneous tissue.

2. Mucous membranes of the respiratory, alimentary, and uro-genital tracts.

3. Serous and synovial membranes.

4. Pia mater, more rarely dura mater.

5. Rarely in brain, spinal cord, salivary glands, mamma, ovaries, etc.

6. Lymphatic glands, spleen, lungs, liver, testes.

7. Bones, especially cancellous bone.

VII. Secondary Changes in Tubercle—

1. Caseation—

This process begins in the centre of the nodule, giving rise to yellowish crumbling masses, composed of fatty debris and degenerated leucocytes. The caseous mass softens, and breaks down, and may lead to the formation of an abscess, then of an ulcer or cavity, as seen in bone, in the lungs, etc. In many cases the caseous matter calcifies.

Causes of Caseation—

(1) May be due to cutting off of the blood supply, for tubercle nodules are non-vascular.

(2) May be due to the direct action of some substance of the nature of a ferment, produced by the agency of the tubercle bacillus,—the cells of the tubercle nodules first becoming hyaline and then caseous. The caseous masses often calcify, forming gritty masses, especially seen in the mesenteric glands.

2. Fibrosis—

This fibroid change in tubercle is frequently seen alone or accompanied by caseation. In the centre of the tubercle there is fatty degeneration, but the leucocytes round the periphery give rise to a reticulum of fibrous tissue, or form a definite capsule of fibrous tissue; the tubercle nodules shrink and leave a fibrous scar with points of fatty degeneration.

This fibroid change is a natural process of cure, and is especially seen where the tubercular process has become chronic, or where obsolescence occurs—*i.e.*, where the tubercle becomes quiescent, surrounded by a dense fibrous capsule. Still the tubercle virus may lie dormant in these masses and under favourable circumstances again become active.

VIII. Causation of Tubercle—

Tuberculosis, as above stated, is due to the action of the tubercle bacillus.

Tubercle Bacillus—

1. Characters.—Consists of minute rods, varying in size from ·3 to ·5 m.; they are rounded at the ends, often curved, and sometimes thread-like, from several bacilli being united together. They contain rounded bodies, probably spores. The tubercle bacillus and its spores are very resistant to external influences, and retain their virulence even when dried.

2. Cultivation.—The tubercle bacillus is somewhat difficult to cultivate outside the body. It grows best on solid blood serum at a temperature of from 30° to 40° C. It does not liquefy jelly, and does not stain easily.

3. Mode of Inoculation.—Tubercle bacilli are found in the sputum, in caseous products, in the urine, etc., of affected animals; and by injecting these, or pure cultivations, into the blood typical tubercular leisons are produced.

In man, inoculation occurs—

(1) By the lungs, through inhaling the bacilli.
(2) By the food.
(3) By direct inoculation, through a wound, an abrasion, etc.

The bacilli are found, often in great numbers, in the tubercular leison, especially within the giant cells where they find a resting place.

IX. Mode of Spread of Tubercle—

1. By continuity of the tissues.
2. By the lymphatics.
3. By the arteries and veins.
4. By direct infection of one part from another.

X. Contagiousness of Tubercle—

Tubercule is not highly contagious, local contact being seldom sufficient for its spread; there must be a previous predisposition, a tubercular diathesis.

XI. Heredity—

1. Though proofs are as yet wanting that tuberculosis can be transmitted directly from the parent to the child, there can be no doubt that the hereditary tendency to tuberculosis is handed down from parent to offspring.

2. Many cases of supposed heredity are due to infection.

3. Some hold that tuberculosis is transmitted through the milk of the mother.

XII. Tuberculosis in Animals—

Bovine Tuberculosis—

1. Definition.—This is a common affection in cattle, and is called "Pearly Disease."

2. Characters.—The tubercle nodules are composed almost entirely of giant cells. The tubercles have a great tendency to calcification.

3. Sites.—It especially affects serous membranes—also the lungs, spleen, lymphatic glands, alimentary tract, liver, etc.

4. Nature and Cause.—Bovine tuberculosis is practically the same as tuberculosis in man. Some hold that the bacilli are larger in size in bovine tuberculosis, but there is not sufficient evidence of this fact. The bacilli are said to occur in milk, the udders of cows being often affected, and thus infection can be carried to man.

Tuberculosis is also seen to occur in monkeys, in horses, and in poultry—the spontaneous disease occurring in fowls being identical, in all respects, with that produced by inoculation from cases of tuberculosis in man.

II.—LUPUS.

I. Definition—

Lupus Vulgaris—one of the granulomata—is a disease of childhood, and is rarely seen after puberty. It is a local affection of the nature of tuberculosis.

II. General Characters—

1. Macroscopic.—Lupus Vulgaris appears as reddish-brown or yellow-red nodules, from the size of a pin's head to a pea. They form jelly-like masses of a semi-translucent appearance, which run together and coalesce. They usually occur on the face, but also on mucous membranes. The nodules are situated in the corium, and are said to spread by means of the lymphatics, but this is doubtful. They often cause great destruction of the part.

2. Microscopic.—The lupus nodules consist of masses of granulation tissue, with giant cells and epithelioid cells similar to those seen in tubercle. The essential character of the leison is an infiltration of the deeper layers of the corium, especially round the blood-vessels, with small round cells. There are many new-formed capillaries, but part of the mass is non-vascular, and thus closely resembles tubercle nodules. On the whole, however, the lupus nodules are more vascular than those due to tuberculosis.

Results—

(1) Lupus nodules may resolve, but they leave permanent fibrous scars—lupus non-excedens.

(2) Some nodules necrose and then produce ulcers—lupus excedens.

III. Nature and Cause—

The exact nature and cause of lupus is as yet undecided, but the weight of evidence points to it being a local tuberculosis of the skin, etc., caused by the agency of a distinct bacillus, identical with or similar to tubercle bacillus.

Lupus erythematosus is an inflammatory disease of the skin, with superficial destruction of the part. It has, in all probability, no connection with lupus vulgaris.

III.—LEPROSY.

(Lepra.)

I. Definition—

Leprosy, also called Elephantiasis Græcorum to distinguish it from Elephantiasis Arabum—due to the agency of filaria sanguinis hominis—is a specific infective disease.

II. Division—

There are two forms—

(1) Tubercular form.
(2) Anæsthetic form.

III. Distribution—

It is met with in Norway, Sweden, Spain, Italy, and tropical countries—India, Egypt, etc.

IV. Sites—

Leprosy chiefly affects the skin and mucous membranes —*e.g.*, of the mouth, larynx, conjunctiva; also nerve trunks, bones, cartilages, and internal organs—causing ulceration and atrophy of the parts affected.

V. General Characters—

1. Tubercular Form.—In this variety there are at first patches of hyperæmia, followed by thickened, firm, red-brown nodules in the skin and mucous membranes. These nodules may ulcerate and cicatrise.

2. Anæsthetic Form.—In this case there are nodular or diffuse swellings round *nerve trunks*, leading to anæsthesia, atrophy of the skin, leaving white, scaly, parchment-like patches—the fingers and toes often falling off; these effects being due to changes in the nerves.

The leison in both forms is characterised by formation of granulation tissue, with small cells like leucocytes, and other large cells—*lepra cells*—multi-nucleated, vacuolated, branched cells.

VI. Cause—

Leprosy is probably due to the action of a distinct bacillus—bacillus lepræ, which is always present in parts affected.

Leprosy Bacillus—

1. Characters.—The Leprosy Bacilli are small, thin, rod-shaped bodies, found in great numbers in the granulation tissue, especially within the lepra cells.

2. Cultivation.—Leprosy Bacillus is somewhat difficult to grow, but has been cultivated on solidified blood serum and cooked egg. The cultivations have not as yet been successfully inoculated, but from the history of the disease there can be little doubt that it is infective and inoculable.

IV.—GLANDERS AND FARCY.

I. DEFINITION—

GLANDERS is the term applied when the mucous membranes of the nose and its prolongations are the site of the leison, spreading thence to the lymphatic glands; hence the name—glanders.

FARCY, on the other hand, is the term applied when the skin and subcutaneous tissues are primarily affected.

Both glanders and farcy, therefore, are two manifestations of one and the same disease, and occur in horses, donkeys, etc. They are communicable from them to man, and can also spread from man to man.

II. DIVISION—The disease may manifest itself in two forms—

1. The Acute form.
2. The Chronic form.

III. GENERAL CHARACTERS—

1. IN HORSES.—The disease usually begins as swellings or nodules of the mucous membranes of the nose, spreading thence, first to the nearest lymphatic glands, then along the respiratory tract to the lungs, and along the alimentary tract. The skin is also affected.

The leisons are inflammatory in character, leading to the formation of diffuse inflammatory formations or to distinct nodules—*Farcy Buds*—characterised by their tendency to early degeneration—caseation—giving rise to abscesses in internal organs, and to ulcers in the skin.

In the chronic form there are large nodules in the skin and subcutaneous tissues, and secondary nodules in muscular septa. Metastatic abscesses occur in internal organs.

2. In Man.—Glanders generally takes the acute form, is inflammatory in character, but nodules, ulcers, and abscesses also occur.

Farcy Buds are best marked in the chronic forms. They consist of nodules from the size of a pin's head to a pea. They tend early to caseate, and in the skin they form ulcers; in internal organs, they lead to abscesses. These nodular leisons consist of masses of granulation tissue with little to distinguish them from the results of severe inflammation except that they soon degenerate and excite suppuration.

IV. Cause—

A Bacillus—the Bacillus of Glanders—has been found in the peri-vascular lymphatics of organs affected.

Bacillus of Glanders—Bacillus Mallei.

1. Characters. — This bacillus is about the same size as the bacillus of tubercle but thicker and broader, forming hollow rods, curved, but not mobile.

2. Cultivation. — The bacillus has been grown on agar-agar and in blood serum, flourishing best at 30° to 40° C. It forms yellow, milk-like patches. It is difficult to stain in the tissues.

3. Inoculation.—The bacillus has been proved to be the cause of glanders, for it can be inoculated in horses, donkeys, guinea pigs, rabbits, etc., and causes the characteristic local leisons of the disease.

V.—SYPHILIS.

I. DEFINITION—

SYPHILIS is a specific infective disease, similar to the other granulomata, owing its origin to a definite micro-organism, probably a bacillus, and characterised by a definite local leison followed by a train of symptoms indicating a general infection of the whole system.

II. **DIVISION.**—Syphilis is divided into—

1. Congenital Syphilis.
2. Acquired Syphilis.

III. GENERAL CHARACTERS—

1. Congenital Syphilis—

1. DEFINITION.—Congenital syphilis—often called inherited syphilis—is syphilis which has been transmitted from parent to offspring, the inoculation occurring either through the placenta or through the sperm, without affecting the mother.

Syphilis due to contact of the fœtus with the maternal structures, at or near the time of birth, belongs to the next form.

2. CHARACTERS.—Congenital syphilis is characterised by—

(1) Eruptions—roseolar in type—in various parts of the body.
(2) Catarrh of nasal mucous membranes.
(3) Superficial ulcerations and mucous tubercles—about the mouth and anus.
(4) Notched and pegged teeth.
(5) Interstitial keratitis.
(6) Chronic inflammations of the ear, etc.

(7) Ulcerations about the palate.
(8) Diseases of joints.
(9) Gumma in various situations.

2. Acquired Syphilis—

1. Definition.—Acquired syphilis is a constitutional disease due to the direct inoculation by a specific virus.

2. Characters.—The leisons induced by the syphilitic virus are usually classed as primary, secondary, and tertiary, but from a pathological point of view these several stages are mere differences of degree.

(1) The Primary Leison—consists of a local sore—a papule or vesicle which becomes hardened and indurated at its base, forming what is known as a hard or *indurated chancre*. The virus next spreads along the lymphatics to the nearest lymphatic glands, which, as a consequence, become hard and shotty to the feel.

(2) The Secondary Leisons—indicate that the syphilitic virus has entered the blood, and has infected the whole system; hence, in this stage—

(*a*) The disease is infective.
(*b*) The leisons produced are symmetrical in distribution.
(*c*) The manifestations are mainly superficial, occurring in the skin, mucous membranes, etc.

(3) The Tertiary Leisons—are characterised—

(*a*) By being asymmetrical in distribution.
(*b*) By being non-infective.
(*c*) By occurring not only in the superficial parts but also in deeper textures and organs—*e.g.*, bone, periosteum, viscera, etc.
(*d*) By the formation of local new growths—gummata—which tend to caseate, to suppurate, and to ulcerate.

IV. Structure—

Morbid Anatomy.—The various changes induced by the syphilitic virus are—

(*a*) In the primary leison—masses of granulation tissue which, however, do not form new connective tissue, and leave no cicatrices.

(*b*) In the secondary stage—they are inflammatory in character—are accompanied by rashes, roseolar eruptions—most common in the skin—by fever, etc.

(*c*) The tertiary form—chronic—has three chief manifestations, viz.:—

(1) Diffuse Interstitial Inflammations.
(2) Gummata.
(3) Syphilitic Endarteritis.

1. Diffuse Interstitial Inflammations—

These are diffuse growths of connective tissue—diffuse hyperplasias—producing, in the parts affected, growths similar to sarcomata; masses of granulation tissue, which, however, differ from ordinary granulation tissue in their great tendency to induration and contraction, and hence causing thickening of mucous membranes; narrowing of tubes, such as larynx, intestine; cicatrisation and contraction in liver, etc. These diffuse growths may occur in any organ or tissue, but are most common—

1. In the liver.
2. In the muscles.
3. In the skin, mucous membranes, submucous tissues—*e.g.*, of larynx, fauces, rectum.
4. In bone, periosteum.

2. Gummata—

1. Definition.—Gummata—Syphilomata—are nodular masses of granulation tissue, especially met with in the

early stages of congenital syphilis, and in the later stages of the acquired form.

2. Structure—

(*a*) *Macroscopic.*—Gummata are rarely seen in the first stages of their formation. They are, however, ill-defined, rounded masses, from the size of a pea to a walnut, and have a reddish-white translucent, gelatinous appearance. In the later stages they become firmer, yellowish and cheesy-looking, and on section have an appearance similar to the section of a horse-chestnut. They are surrounded by a distinct fibrous capsule, but they cannot be enucleated for they infiltrate the surrounding textures. They are especially well seen in the liver and in the brain. In still later stages they are found to have undergone complete caseous necrosis.

(*b*) *Microscopic*—

1. In the early stages gummata consist of a mass of granulation tissue infiltrating the affected part.

2. Later, three more or less definite zones can be distinguished—

(1) A central zone of caseous and fatty debris.
(2) Outside this, an area of fibrous tissue consisting of cells embedded in a fibrillar matrix.
(3) A third zone of small-celled infiltrations.

3. Sites—Gummata occur—

(1) In the skin, subcutaneous submucous tissues—especially of the pharynx, palate, tongue, larynx.
(2) In the muscles, fascia, bones.
(3) In the membranes of the brain.
(4) In the liver, spleen, testicle, kidney, rarely in the lungs.

4. Changes in Gummata—

(1) Resolution and absorption.
(2) Softening, necrosis, leading to the formation of cysts.
(3) Induration.

Caseation is the characteristic change in gummata, it is a coagulative necrosis due to changes in the blood-vessels.

3. Syphilitic Endarteritis—Endarteritis Obliterans. —This is the third characteristic change due to syphilis and consists in an irregular thickening of the walls of the arteries, leading to narrowing and occlusion of their lumen, and subsequently to thrombosis. The smaller arteries and veins are chiefly affected, the change commencing in the inner coat and consisting of a formation of granulation tissue, composed of fibrous tissue, of small round cells, and of spindle-shaped cells. The outer coat becomes ultimately affected in a similar manner.

Atheroma and amyloid degenerations are a frequent consequence of the syphilitic leison.

V. Cause—

Nothing is certainly known as to the specific virus of syphilis. It is probably of the nature of a bacillus, several of which have been described, but their relations to the characteristic leisons have not yet been determined.

The virus exists in the primary chancre, in all the secondary leisons; the normal secretions however—mucus, saliva, etc., are not infective.

The mode of entrance of the virus is probably through abraded surfaces of the mucous membranes and the skin, and is thence carried to the lymphatics, lymphatic glands, and the blood.

VI.—ACTINOMYCOSIS.

(ἀκτις—a ray.)

I. Definition—

Actinomycosis is a disease especially affecting cattle, but is also found in man, and is due to the action of a definite fungus—the ray fungus—actinomycoses.

II. General Characters—

Macroscopic.

1. In Cattle.—The disease usually begins in the lower jaw, and causes sarcomatous-like growths—bulky tumour-like masses—connected with the bone. The bone is softened, eroded, leading to the formation of an abscess. The tongue is also affected, hard indurated nodules being produced which give a gritty, woody feel; hence the name, Woody Tongue. The virus is carried by grain. It does not affect carnivora.

Sites of the leison.

1. Jaws—tongue—neck—glands beneath the jaw.
2. Larynx—lung—alimentary tract.

2. In Man.—The disease also affects the jaw, and leads, as in cattle, to the formation of nodular masses, of deep-seated abscesses—retro-pharyngeal—in the region of the spine, etc.

Nodules also occur in the lungs—patches of chronic induration, abscesses, and cavities.

The liver, intestine, the kidneys, are also affected.

The masses tend to suppurate, not to caseate.

III. Structure—

Microscopic.

The nodules are seen to consist of masses of granulation tissue. They have a sponge-like arrangement, and contain

small yellowish granules. They have a structure very similar to that found in tubercle, being composed of—

1. The fungus—ray fungus in the centre.
2. Round this there are the giant cells.
3. Epithelioid cells.
4. Masses of granulation tissue.

IV. MODE OF INOCULATION—

1. Through the mouth by carious teeth, etc.
2. By the respiratory tract.
3. Through the intestinal tract.
4. In many cases the mode of inoculation is unknown.

V. CAUSE—

Actinomycosis is due to a fungus — the ray fungus — which is found in all the leisons.

Ray Fungus—

1. CHARACTERS.—The exact botanical position of the ray fungus is not known. By some it is regarded as a mycelium, by others as a leptothrix or cladothrix.

2. STRUCTURE.—The fungus consists of short threads or rods, often club-shaped, dotted, branched, sometimes calcified, arranged in a radiating manner round a common centre, composed of fine filamentous fibres. They form the globular masses already spoken of. The central part of the mass is the living cladothrix, the rays are degenerated filaments.

3. CULTIVATION.—Actinomycoses has been cultivated but not with uniform success. It has also been inoculated, but here also, the results have not been altogether very satisfactory.

II.—BLASTOMYCETÆ.

(Yeasts.)

I. DEFINITION—

The Blastomycetes—yeasts—sprouting fungi—are the chief agents in the process of fermentation and are only important in that respect.

II. GENERAL CHARACTERS—

The yeasts are small, round or oval, granular cells, often vacuolated, sometimes forming branching chains.

Few of them are parasitic, most are saprophytes.

III. REPRODUCTION—

They grow by a process of gemmation—buds growing out from the cells, enlarging, and either remaining attached or falling off. Sometimes, especially when cultivated artificially, they form spores.

IV. EXAMPLES—

1. SACCHAROMYCES CEREVISIÆ—Beer yeast—common cause of fermentation.

2. SACCHAROMYCES MYCODERMA—Vinegar plant.

3. SACCHAROMYCES ALBICANS—Oidium albicans, the parasite of thrush.

THRUSH.

THRUSH—Aphthous Stomatitis—consists of milk-white or grey-looking, curdy, adherent patches on the mucous membrane of mouth and pharynx of weakly children that

are suckling, and of adults reduced by diseases, such as typhoid, etc.

The patches consist of epithelium united into a membrane by a fungus—the *oidium albicans*—consisting of twisted filaments, often branched, composed of long cells joined end to end and constricted at the joints. By some this fungus is regarded as a yeast, by others as a mould.

III.—HYPHOMYCETÆ.

(Moulds.)

I. Definition—

The Hyphomycetæ—moulds or filamentous fungi—are formed of a single row of cells placed end to end.

II. Characters—

The moulds consist of filaments—hyphæ—which may be single or may cross each other in all directions. These filaments are of two kinds—

1. The Nutrient Hyphæ—which form a mycelium which corresponds to the root. They grow into the nutrient medium, and extracts nutriment therefrom.

2. The Reproductive Hyphæ—which spring upwards from the mycelium and carry the conidia—organs of fructification.

The spores are rounded or oval, coloured or colourless, and motionless.

III. Examples—

1. Non-Pathogenic Moulds.

(1) Penicillium Glaucum—the blue mould seen in decomposing food kept in moist places.

(2) Aspergillus Glaucus—the common mould seen in rotten fruits, wood, etc.

(3) Mucor Mucedo—the white mould seen on horse-dung, etc.

2. Pathogenic Moulds.

(1) Achorion Schœnleinii—the cause of Favus.

(2) Trichophyton Tonsurans — the cause of Ringworm, etc.

(3) Microsporon Furfur — the cause of Pityriasis Versicolor.

I.—FAVUS.

Favus. — This disease occurs as light yellow crusts, chiefly on the hairy part of the scalp. These crusts consist of the fungus *Achorion Schœnleinii*, which invades the roots of the hairs, growing in the epithelium of the hair follicles, and penetrating into the shaft of the hair.

The parasite consists of an unjointed, branching, densely interwoven network of filaments, often with oval spores. It has the smell of mice.

II.—TINEA TONSURANS.

Tinea Tonsurans—Ringworm—which chiefly affects the hair of the scalp, Tinea Sycosis—attacking the beard, and Tinea Circinata—growing in the epidermis of other parts of the body, are due to the fungus *Trichophyton Tonsurans*.

This parasite consists of hyphæ and spores, which grow in the epidermis of the roots and deep parts of the shafts of hairs, and give rise to great irritation and even suppuration.

III.—PITYRIASIS VERSICOLOR.

Pityriasis Versicolor.—This is a disease in which there are yellowish or dark brown, scaly patches over the trunk. The patches consist of epidermic scales and the fungus *Microsporon Furfur*, composed of jointed threads and spores.

IV.—MADURA FOOT.

This is a disease met with in parts of India, causing swelling of the feet, due to cavities and tubercles in the structures beneath the skin.

The sinuses contain pus in which are the spores and filaments of a fungus — the *Chionyphe Carteri* — which is supposed to be the cause of the disease, but this is doubtful.

The exact botanical position of this fungus is as yet undecided.

INDEX.

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2